BRAIN.

PARTS III and IV, VOL. 39.

THE POSSIBLE FUNCTIONS OF THE CEREBROSPINAL FLUID.[1]

BY PROFESSOR W. D. HALLIBURTON, M.D., F.R.S.

So much has within recent years been written about the cerebrospinal fluid that I must apologize for my want of originality in selecting that subject for this address. But however hackneyed the subject, we are still far from possessing complete knowledge as to the functions and significance of the fluid. From the pathological as well as from the physiological point of view, we at least know that this remarkable fluid is one of the greatest importance, and its careful examination in many diseases has yielded results of inestimable value. During the last few years it is a subject at which I have myself worked, and I have been fortunate in securing as my collaborator such a skilful and accurate experimenter as my colleague Professor W. E. Dixon.

One cannot work at any subject for any length of time without speculating beyond actually observed data. It is the safest rule to keep ideas of this nature to oneself, but there are some occasions when it is permissible to speak rather more freely; this is one of them. I happen to be the unworthy occupant of a presidential chair, and for an hour or less I shall be free from contradiction and criticism, so I propose to inflict upon you during that time some speculations, and have only safeguarded myself in the title of this address by alluding to the functions of the fluid as possible rather than probable. As my colleague Professor Dixon is away on War service, I have had no opportunity of discussing the matter with him, so that if I say anything that is wildly improbable he at any rate is free from responsibility.

I shall dwell on the subject from the physiological rather than from

[1] Presidential Address delivered before the Neurological Section of the Royal Society of Medicine on October 26, 1916.

the pathological standpoint, but I trust that what I have to say will be found of interest to the pathologist and the clinical observer.

May I, after this preface, proceed to another chapter, which is also of a prefatory character, and state as shortly as I can the present state of our knowledge regarding the characters, composition and fate of the fluid. The fluid, as you know, is, under normal conditions, as clear as water and of low specific gravity; it contains in solution inorganic salts similar to those in the blood-plasma, a trace of coagulable protein, and a certain amount of a reducing substance which has now been definitely proved to be glucose; it is practically free from formed elements. Under various abnormal conditions the protein matter may be largely increased, or substances other than the usual ones may be added to it, such as cholesterol and choline (or a choline-like substance). It may further have added to it various kinds of cellular structures, the differentiation between which is a valuable aid in diagnosis, and in other cases parasites of different sorts may be found. The fluid is primarily formed by the secreting cells which are most prominently found covering the choroid plexuses in the cerebral ventricles, so that this structure may be appropriately designated, as Mott first suggested, the choroid gland. The fluid is normally present at a certain pressure, and this pressure is not the result of arterial pressure passively transmitted to the fluid. The cerebrospinal pressure, it is true, may be affected by changes in the arterial and venous pressures, but it is not dependent on them; and it may and often does vary quite independently of these. The true cerebrospinal pressure is the result of the secretory pressure of the choroid epithelium cells. In other words, the craniospinal contents cannot any longer be regarded as a fixed quantity without the power of expanding or contracting in volume.

The rate of flow and the pressure of the fluid can be readily investigated in animals by placing a cannula in the subcerebellar cisterna and connecting it with the necessary apparatus. It is then found that there are three groups of substances which promote the flow and increase the pressure independently of those which affect it secondarily by altering the blood-pressure. The first group consists of excess of carbon dioxide (or lack of oxygen) in the blood, as in asphyxia, and drugs which interfere with respiration. The second group is that of the volatile anæsthetics, which may act by interfering with the respiration or by altering the physical conditions of secretion. The third group is specific, and consists of an extract of the choroid gland, or of the brain. The former

is the more powerful. The chemical nature of the hormone in this extract is uncertain, but it is probably some product of nerve metabolism, which, arising in the brain, passes to the choroid plexuses, accumulates there, and stimulates the secreting cells to activity; it cannot be discovered in the normal secretion, but in cases of general paralysis and brain softening—conditions in which catabolic processes are excessive—it can be recognized (by physiological tests) in the fluid itself. The choroid plexuses are abundantly provided with nerves, but there is no evidence that these are secretory in nature; indeed the evidence derived from experiments with atropine and similar alkaloids points in the opposite direction. The hormone, whatever its nature, acts probably, not on nerves, but on the secreting cells directly.

There is no doubt that this fluid is being continually formed, and the next question is—What becomes of it? Large quantities of a neutral fluid, such as physiological saline solution, disappear within a few minutes when introduced into the craniovertebral cavity, and the course taken by such fluids, and presumably also of the normal cerebrospinal fluid, can be traced by adding some substance which can be easily recognized by its colour, or by chemical tests, or by its physiological action. Using such methods, it has been demonstrated that the exit is by the blood-stream, and not by the lymph channels of the nerves, which was formerly thought to be the case by a number of French observers.

If such substances are readily diffusible the speed with which they appear in the blood is very remarkable, especially if they are introduced into the subcerebellar region. An injection, for instance, of adrenaline, nicotine, or atropine produces typical physiological actions within a few seconds, in fact, almost as rapidly as if the injection had been made into the venous circulation direct.

On the other hand, substances which are not readily diffusible (such as commercial peptone) do not produce their characteristic effects when they are introduced into the cerebrospinal fluid, so that one of the older theories that actual valved orifices exist leading into the large veins at the base of the brain must be abandoned.

The diffusion process is most rapid in the subcerebellar district, but is extremely slow in the spinal, especially the lower spinal region.[1] It probably occurs into the venous sinuses by the microscopic arachnoid

[1] This is tacitly accepted by those who produce spinal anæsthesia by the injection intrathecally of substances of the cocaine group, for they recognize the danger of absorption if the drug reaches the medullary region.

villi described by Weed. There is also a possibility that in addition to this, transference may occur through the thin walls of the blood-vessels within the central nervous system, for, as Mott has pointed out, contact of these vessels with the cerebrospinal fluid is maintained throughout their extent by the perivascular spaces which are continuous with the subarachnoid cavity. Diffusion in the opposite direction from blood to cerebrospinal fluid does not occur except in an almost negligible degree in the case of a few drugs, such as alcohol and urethane.

But in addition to this there is another and minor communication between the fluid and the other parts of the body. Dixon and I found that dyes added to the fluid travel along the course of certain cranial nerves, and this is especially true for the olfactory nerve. This is not the case for the spinal nerves; no dye can be detected in their sheaths outside the spinal canal, and no dye is discernible in the lymph of the thoracic duct. I have spoken of this cranial (olfactory) outlet as a minor one, but clinical experience has shown that it is not a negligible one. For this loophole affords an opportunity for the entry of infective agents, as Flexner has shown in the causation of infective polio-myelitis.

Such then is a brief and I fear imperfect summary of the present state of our knowledge, and I propose now to pass to the main object of my address, which is to discuss the meaning and functions of the fluid we are dealing with.

In the first place it can hardly be doubted that the presence of fluid within and around the structures of the central nervous system fulfils certain mechanical functions of support and pressure. That, however, does not explain why ordinary lymph would not do just as well, as it does in the majority of other organs. The relationships, however, of the brain and cord within a closed cavity are peculiar, and it may be that ordinary lymph is here insufficient to maintain a more or less constant pressure. For the pressure of ordinary lymph is wholly dependent on blood-pressure; here, in addition, we have an independent pressure, namely, the secretory pressure of the choroidal secreting cells, and it is therefore quite possible that this may come into play in maintaining, equalizing, and adjusting those pressure relationships which are most advantageous for the well-being and function of the brain and cord. It is further quite possible that the ameliorative effects not infrequently noticed as a result of withdrawal of the cerebrospinal fluid by lumbar puncture may be due to the relief of undue pressure. But such

considerations offer no explanation of the peculiar composition of the fluid. Its characteristic chemical composition must have a deeper meaning.

It is very common to speak of the cerebrospinal fluid as the lymph of the brain. Let us now examine this phrase more fully, and see if it has any scientific accuracy.

In the first place we must be quite clear as to what lymph is, and what it does. It is a fluid which exudes through the thin walls of the blood capillaries. Whether lymph formation depends solely on physical conditions (filtration and osmosis), or whether in addition we have as a factor a secretory activity of the vascular lining membrane, is an interesting physiological problem, which need not concern us now. The leakage fluid resembles blood-plasma in its composition, except that it is comparatively poor in protein material, the diffusion of which through membranes is so difficult. This lymph, when formed, acts as the intermediary or middle-man between the blood and the tissue elements, conveying to the latter on the one hand the oxygen and nutritive substances they need; and on the other hand it is into the lymph primarily that the tissues pour the waste products of their activity, and thus these are started on their journey to the organs of excretion (lungs, kidneys, &c.). The very essence of a lymph is that it should be in free communication, except for an intervening membrane, with the blood-stream, and that this membrane should be equally permeable to water and other substances in both directions. The arrangement of the perivascular and perineuronal spaces, filled as they are with cerebrospinal fluid, certainly resembles that of a lymphatic system, and therefore it is not surprising that the idea has caught on that cerebrospinal fluid plays in the central nervous system the rôle of lymph. The peripheral nerves have a true lymphatic system analogous to that found in other organs, but anatomists are far from unanimous on the question whether the central nervous system possesses real lymphatic channels apart from the system of intercommunicating spaces occupied by cerebrospinal fluid. The mere difficulty of discovering true lymph-vessels does not indeed finally negative their existence, but until they are satisfactorily demonstrated we may provisionally assume that they are absent. If, then, cerebrospinal fluid is the only fluid which actually comes into contact with the tissue elements of the brain and cord, it necessarily follows that it must play the part played by lymph in other districts of the body; it must be for example the intermediary medium which is traversed by the oxygen on its way from blood to the tissue elements;

and oxygen we know is essential for the continuance of nervous life and energy. Furthermore it must be the vehicle by which other nutriment reaches the cells and fibres of nervous tissue. The next point is, have we any evidence that the products of nerve katabolism pass into the fluid from the tissue elements as they do into ordinary lymph? If it were only possible to analyse and compare the composition of the fluid before it enters the perineuronal spaces, and after it leaves them, it would be possible to answer this question authoritatively, but in the absence of such proof one can only argue from probabilities. Seeing that the fluid is the only one available for the purpose, an affirmative answer seems inevitable, and this is supported by the fact that cerebrospinal fluid is rich at any rate in one waste product—namely, carbon dioxide.

The expression "lymph of the brain" is therefore so far justifiable. But we have absolutely no proof that the cerebrospinal fluid is in part an exudation from the blood, and it is in the origin of the fluid that the analogy between lymph and cerebrospinal fluid breaks down. Whatever views we may hold as to whether a secretory factor comes into play in the production of ordinary lymph (and, at the best, such a factor can only be an insignificant one), we can have no hesitation in proclaiming that cerebrospinal fluid is a true secretion arising in a definite glandular structure. Such a fluid must obviously be the best one for maintaining normal life in the nerve cells, and the whole lining membrane of the spaces in which it resides appears to co-operate with the choroid gland in maintaining its constancy of composition, and to militate against the escape into it of substances from the blood-stream, such as drugs or poisons which would be foreign to the fluid or harmful to the delicate and sensitive structures which it bathes.

We have seen that the essential feature of a true lymph is the free interchange between it and the blood in both directions. This essential character is lacking in the cerebrospinal fluid: in all probability the lining membrane of the cerebrospinal spaces is permeable to substances passing from it into the blood, but it appears to be impermeable (except for oxygen) in the direction from the blood to the fluid. The nutritive materials the fluid contains appear to be formed in the choroid gland, and not to be merely exuded from the blood-stream; otherwise one cannot explain why the protein it contains is not similar to that in the blood or in exudations (lymph) formed from the blood.

If, as Weed has suggested, cerebrospinal fluid is partly formed, as ordinary lymph is, by exudation from the blood in the perivascular spaces,

it is difficult to understand why readily diffusible drugs and poisons do not escape readily into the cerebrospinal fluid as they do into ordinary lymph. The difficulty is quite intelligible when we regard the choroidal epithelium as a stalwart barrier of cells which keeps back these materials, and only allows its own normal secretion to escape. This useful work would be undermined and frustrated if the general lining were easily permeable to foreign substances.

Camus found that barium chloride, which is a very active poison to the central nervous system, will kill a rabbit of 2 kgr. weight when $\frac{1}{10}$ mgr. is introduced into the subarachnoid space, whereas the lethal dose is one thousand times greater when this salt is given subcutaneously. It is well known that anaphylaxis can be produced by smaller doses of proteins administered into the brain than when given elsewhere. The use of salvarsan in locomotor ataxy and similar late syphilitic affections *via* the cerebrospinal fluid has been abandoned, as it is fatal not only to the syphilitic organisms, but also to the patient. It is further known that salvarsan and its homologues are of little or no use in tabes and general paralysis when it is given by the ordinary channels, for in these later manifestations of syphilis the baneful spirochæte has got into a harbour of refuge (which we may speak of as extravascular) beyond the reach of the poison.

May I add in way of parenthesis how very desirable it is that chemists and pharmacologists should apply themselves to the solving of this difficulty. The future treatment of tabes and allied conditions should aim at the discovery of some blander arsenic compound which could be introduced straight into the cerebrospinal fluid and kill the syphilitic organism in its lair without at the same time slaying the host who harbours it.

Is not the very simplicity of the normal fluid suggestive? I remember when I was a student that normal or physiological saline solution was regarded as a physiological fetish, for surgeons continued to use sponges (usually dirty ones at that time) soaked in water during operations. What a change has now come over the spirit of their dream! They have realized that water, even clean water, is a protoplasmic poison, and that osmosis is a real force and not a negligible phenomenon. I remember, at about the same date, witnessing, and in a humble measure assisting, Dr. Sydney Ringer in his epoch-making work on the effects of saline mixtures on living structures. The attitude of the profession generally in those days was a tolerant one, just the sort of attitude adopted by a grandfather indulgently watching his descendants

playing with their toys. But who has not heard of Ringer's solution to-day? Who can write a paper on almost any physiological or pathological subject without mentioning it or alluding to the part it has played in his investigations?

Ringer's fluid is the ideal physiological salt solution; its saline constituents are present in the same proportion as they occur in the natural body fluids, and in that way the normal osmotic pressure is maintained when living structures are bathed in it. Ringer's original solution resembled frog's blood in its saline composition, but since then several modifications have been introduced. The most important of these is the one we owe to Locke, who has altered the proportion of the various salts (increasing, for example, the amount of sodium chloride from 0·6 to 0·9 per cent.), so that the fluid may be employed with success on the living tissues of the mammal. Locke has also added a small proportion of the sugar glucose, and when in use it is kept saturated with oxygen. It is well known that in such a fluid living structures can be preserved in a living condition for hours, or even days and weeks. Locke's most striking results have been obtained with the mammalian heart: this can be kept beating for prolonged periods after its complete isolation from the body if it is perfused with the oxygenated fluid. The salts supply the normal stimulus to the cardiac fibres and maintain their integrity; the sugar acts as a source of energy and is consumed as activity continues.

Ringer's fluid, so compounded in the laboratory, is somewhat more than thirty years old. But in reality it is as old as the hills, or rather as man himself. Just as the Venus of Milo existed potentially from past ages in the block of marble from which it was ultimately hewn, so does Ringer's fluid exist in the blood and lymph, although its simple composition is obscured there by admixture with the blood proteins and corpuscles.

Here may I introduce another parenthesis and allude to another physiological puzzle, and that is the significance of the large amount of protein in the blood plasma and lymph. The meaning and use of the blood corpuscles we understand, or at least we think we do, but the meaning of so large a quantity of protein in the blood fluid and the part it plays in nutrition is still hidden from us. The amount of protein necessary for the repair of the tissues is not great, and the modern doctrine of maintenance and growth is that the tissues help themselves, not from proteins directly, but from the various amino-acids which are the result of protein cleavage. The great protein store may be another

instance of the prodigality of Nature in providing a large margin for conditions of weakness and stress. However this may be, the presence of protein is not necessary, or it may even be harmful when it is added to Ringer's or Locke's solutions in experiments with perfused organs.

But to resume the thread of our argument. Thanks to Ringer some thirty years ago, physiologists were provided with a suitable fluid for experimental work, but the choroidal epithelium possessed the secret of extracting it from the blood for untold ages before Ringer was born. For what after all is cerebrospinal fluid but Locke's modification of Ringer's solution?

The view I have been led to take is—the nervous mechanism being so sensitive, so easily influenced by anything unusual—that therefore the neurons must be bathed in an ideal physiological saline solution to maintain their osmotic equilibrium; the trace of protein it contains is probably quite sufficient for nutritive processes, and is no doubt the kind of protein particularly suited to repair the small amount of wear and tear which is the result of nervous action. The sugar, just as in Locke's solution, would serve for a supply of energy. The choroidal epithelium, in its wise choice of a suitable circumambient medium for the neurons, is really exercising a protective function. In order to keep out harmful proteins (toxins and the like), the comparatively harmless ones are kept back also, almost completely; all share the same process of exclusion. This protective action applies in addition to the majority of soluble drugs; this, as we have seen, may operate so as to be detrimental in diseased conditions. But we can hardly expect discrimination on the part of the epithelial secreting cells. The non-access of metallic and other poisons to the nervous elements is such a *sine quâ non* for their health, that during those periods when such substances are given for the relief of disease, or the slaughtering of parasites, the choroidal cells are unable to change their habits and do not allow the drugs to get through.

Such I believe then is the real significance of the simple composition of this remarkable secretion. But before I sit down I must allude, in conclusion, to some other possible additional functions which the fluid may exercise.

The late Dr. Gaskell approached the question from the embryological and developmental point of view; he held that the neural tube represents an ancestral digestive canal, and those who adopt Gaskell's hypothesis might conceivably argue that the cerebrospinal fluid is the representative of a primitive digestive juice, and is secreted by an organ

which was formerly a digestive gland. However interesting such speculations may be, one can hardly suppose that anyone could seriously urge that the cerebrospinal fluid retains any such functions to-day.

Dendy is one of many comparative anatomists who have not accepted Gaskell's views, and he has suggested that the important function fulfilled by the choroid plexuses is that of an intracerebral gill, and is concerned in respiration. The structure of the plexuses is certainly gill-like, but it is always unwise to argue from mere anatomical resemblances. Mott, who analysed the gases of cerebrospinal fluid, found the quantity of carbon dioxide there very high (about 60 per cent.), and there is, therefore, something to be said in favour of the respiration hypothesis. It is quite possible that the choroidal epithelium may allow the escape of or even actively excrete this catabolic product, and it is unnecessary to point out how severe a poison any undue accumulation of carbon dioxide is in the central nervous system. Coupled with this fact is another I have previously mentioned—namely, that carbon dioxide is one of the most potent means of promoting a flow of the fluid. Just as urea is the best diuretic, and bile the best cholagogue, so carbon dioxide is one of the most powerful cerebral lymphagogues.

One further point and I have done. The ependyma is lined by ciliated epithelium. Some doubt exists, I believe, as to whether the cilia are functionally active. But if we admit they are, the question arises—In which direction do they act? This is a question which is hardly susceptible of actual observation in man or the higher animals, but in the Amnocates, which Professor Dendy has so largely used in his investigation, he has brought forward evidence which shows that the movement is in a forward direction. I know it is not always wise to draw conclusions from such lowly animals, and apply them without reserve to the higher ones. There are certain cases where such deductions are quite impossible, for instance, in some of the humble fishes the central canal of the spinal cord does not terminate blindly at its posterior extremity, but opens out by an orifice into the surrounding tissues.

In this case one can only speculate, and the rapidity of absorption of the fluid in the forward regions in comparison with that in the spinal district appears to favour Dendy's surmise. The cilia, if they are active, would no doubt further the flow of fluid from the cord region to the large veins at the base of the brain, where it so readily leaves the subarachnoid space by entering the venous blood-stream.

There are doubtless other problems in connection with the cerebrospinal fluid that demand solution, but my task is over. My main object has been to present the thesis in which I feel there is, at any rate, some truth—namely, to regard the cerebrospinal fluid as the perfect physiological medium, more perfect doubtless than the artificial fluids we can make in the laboratory, but in its essential features closely resembling those associated with the names of Ringer and Locke.

These are days of specialization, but however necessary specialization in medical science may be it has its attendant evils. Particularly regrettable is the divorce between those who pursue their investigations by the bedside and those who work in the laboratory. Neurology specially is a branch of our science in which an attempt should be made to bring about a closer *rapprochement* between the two sets of workers. Speaking personally I have derived inestimable benefit from hearing here the clinical side of the subject. Those responsible for the management of this Section of the Royal Society of Medicine have realized this, and in having elected this year a President who is a laboratory worker they have shown that they see the value of the *entente* I have alluded to; and I can only trust that my year of office may not be detrimental to those who form the larger contingent in the Neurological Section—namely, those who pursue more specially the study of disease by the bedside.

INTRACRANIAL ANEURYSMS.

BY E. G. FEARNSIDES, M.D.CANTAB., F.R.C.P.

Assistant Physician to the Hospital for Epilepsy, Paralysis and other Diseases of the Nervous System, Maida Vale; late Medical Registrar to the London Hospital; Beit Memorial Research Fellow.

PAGE

INTRODUCTION 224

CHAPTER I.—HISTORY 225

CHAPTER II.—THE PATHOLOGY OF INTRACRANIAL ANEURYSMS 231

CHAPTER III.—THE CLINICAL MANIFESTATIONS ASSOCIATED WITH INTRACRANIAL ANEURYSMS OF NON-EMBOLIC ORIGIN 243

CHAPTER IV.—THE CLINICAL MANIFESTATIONS ASSOCIATED WITH INTRACRANIAL ANEURYSMS OF EMBOLIC ORIGIN 276

CHAPTER V.—SUMMARY OF CONCLUSIONS 281

LITERATURE 283

APPENDIX OF CASES 284

THE presence of a saccular aneurysm on one of the basal cerebral arteries at necropsy is one of the commonest pathological findings. Between 1907 and 1913 in the Pathological Institute of the London Hospital 7,924 *post-mortem* examinations of the body were made, in 5,432 of which an examination of the head was permitted, and saccular cerebral aneurysms were discovered in 44 cases. Osler [17] in his first 800 autopsies in Montreal found 12 instances, whilst Newton Pitt [16] in the dead-house of Guy's Hospital between the years 1869 and 1888 found 23 instances in nearly 9,000 examinations. In the past the chief causes of the formation of these aneurysms have been found in (i) embolism and (ii) syphilitic arteritis. In a recent paper entitled "Alterations in Arterial Structure and their Relation to Syphilis," H. M. Turnbull [24] has reviewed our knowledge as to the causations of aneurysms in general and of the morbid changes in the walls of the affected vessels. This paper, which is based upon the findings in the *post-mortem* room of the London Hospital during the years 1907 to 1913 inclusive, proves that the commonly accepted views as to the relation of intracranial aneurysm to an infection with the *Spirochæta pallida* are in need of revision.

Chapter I.—History.

The first definite account which I have been able to find of any intracranial aneurysm was published by Biumi of Milan in 1765, and was reprinted by Sandifort at Leyden in 1778; in this case an aneurysm of the internal carotid artery was found in a female of advanced age who had suffered from several severe rheumatic attacks. In 1814 Blackall in his work on "Dropsy" described an aneurysm of the basilar artery in a young female which had ruptured and produced fatal coma. Hodgson in the following year republished this case in detail and also described a second in which an aneurysm of the anterior cerebral artery, completely filled with blood-clot, was found at the *post-mortem* examination of the brain of an incurable lunatic who had been an in-patient for seventeen years at Bethlem Hospital; he referred to a third case, which had come under his notice, of a ruptured aneurysm of the size of a cherry on one of the cerebral arteries. In 1825 Spurgin of Saffron Walden published a case of aneurysm of the anterior cerebral artery, which occurred in a man of 57 who was suddenly seized with apoplectic symptoms; after a partial recovery and relapse he had a third fit in which he died. The aneurysm was about the size of a hazel-nut and had ruptured. In 1826 Serres published two cases; the first occurred in a man of 59 who died suddenly during convalescence from pneumonia; here, a ruptured aneurysm, of the size of a hen's egg, was found on the basilar artery. The second in a woman aged 53 who had suffered from epileptic seizures and was paralysed on the left side, where a bilocular ruptured aneurysm of the anterior communicating artery was found. Since this time records of cases of intracranial aneurysms are found scattered through medical literature. In 1834 Nebel first collected together and analysed the clinical findings in thirteen cases, and in 1836 Stumpff wrote a graduation thesis on fifteen cases. Brinton [5] in 1852, after describing a case of unruptured aneurysm of the anterior communicating artery, appended a table in which he gave statistical details of forty-nine cases. Gull [11] in 1859 entered more fully into the symptoms caused by aneurysms of the cerebral vessels and endeavoured to establish means by which they might be diagnosed during life, but concluded that "although we may from the circumstances sometimes suspect the presence of aneurysm within the cranium, we have at the best no symptoms upon which to ground more than a probable diagnosis."

The series of papers which Lebert [15] published in 1866 are

classical contributions to our knowledge of cerebral aneurysms. Lebert collected the records of 86 cases. He found that 47 of these had occurred on the carotid system of the arteries at the base of the brain and thirty-eight on the vertebral system, and that the incidence had been greatest on the middle cerebral arteries. Fifty-two of his cases had occurred in males and thirty in females; the arteries on the two halves of the body had been affected equally, except in the case of the internal carotid and posterior communicating arteries, where there was a preponderance on the left side. He first noted the frequency of the association of intracranial aneurysm with cardiac disease, but did not trace any causative relationship between these conditions. He gave an extremely lucid account of the clinical manifestations which had been observed in cases of cerebral aneurysm, and stated that in two-thirds of the cases of aneurysm of the middle cerebral arteries, and in 56 per cent. of all cases, rupture of the aneurysm had preceded and led to death. He correlated the common latency and exacerbation of symptoms with the liability of the aneurysms to partial ruptures. As to causes of cerebral aneurysms he considered mechanical, traumatic, and histological factors; in his opinion the majority were due to a "parenchymatous arteritis." "Calcifying atheroma is rarely found in cases of cerebral aneurysm and is not an important cause of their formation." He considered that the few cases which had occurred in patients giving a history of chronic alcoholism or of syphilitic infection did not support the hypothesis that these conditions bear any direct relationship to the formation of cerebral aneurysms. From his researches he was led to believe "that in a number of cases a definite diagnosis of cerebral aneurysm is possible."

Church [8] in 1870 first traced the causative relationship between the formation of intracranial aneurysms and vegetative endocarditis. Church was able to collect 13 cases, four of his own and nine from the literature, of cerebral aneurysms occurring in patients under 20 years of age due to emboli arising from endocarditic vegetations; these occurred on the right middle cerebral artery in 3 cases, on the left middle cerebral artery in 2 cases, on the basilar artery in 2 cases, on the posterior communicating artery in 2 cases, on the posterior cerebral in 1 case, on the right anterior cerebral in 1 case, on the left anterior cerebral in 1 case and on the left internal carotid in 1 case. At the end of his paper he stated that "further research has convinced me that intracranial aneurysms in the young are nearly always due to embolism."

Bartholow [1] in 1872, after reporting one new personally observed

and six newly collected cases, again reviewed the literature. From his studies of the records of 114 cases he concluded "there is much reason for believing that syphilis is the chief cause of the formation of intracranial aneurysms" and that after syphilitic aneurysms in order of frequency come those due to embolism from cardiac disease.

Ponfick [21] in 1873 clearly demonstrated the causative relationship between emboli and aneurysmal formation. The detailed description of the organs in his cases proves that all were examples of progressive, ulcerative endocarditis, but in the article of his paper he failed to recognize the infective nature of the emboli and attempted to explain the formation of the aneurysms on a mechanical basis.

Peacock [20] in 1876 analysed the pathological findings in cases of cerebral aneurysm. He reported 3 new cases and collected 86 from literature; he found that the internal carotid arteries had been affected in 12 cases, the anterior cerebral arteries in 4 cases, the anterior communicating arteries in 5 cases, the middle cerebral arteries in 27 cases, the vertebral arteries in 5 cases, the basilar arteries in 22 cases, the cerebellar arteries in three cases, and the posterior cerebral arteries in 6 cases. Death occurred from rupture of the sac in 17 of the 27 cases of aneurysm of the middle cerebral artery and in 14 of the 22 cases of aneurysm of the basilar artery. Of these aneurysms, 40 occurred on the left side and only 31 on the right, and 37 occurred in males and 43 in females. In Peacock's list no case of aneurysm of the posterior communicating artery appears, but, as Peacock notes, Bartholow in 1872 had already reported 8 such aneurysms. As to causation he remarks that "in some cases the disease seems to have orginated in embolism" . . . "not infrequently the aneurysms seem to be caused by syphilis" . . . "the other cerebral arteries are often extensively diseased in cases of aneurysm, being thickened, atheromatous, and indurated, but in some cases the disease of the vessels is said to have been limited to the immediate seat of the aneurysm."

Eppinger [9], whose classical work on the pathology of aneurysms in general appeared in 1887, insisted that in the small and smallest arteries, like those forming the circle of Willis and its branches, many aneurysms have a congenital origin and are due to an inborn defect of the elastic properties of the arterial wall; in others congenital defects with subsequent degenerative or atheromatous processes complicating the defect are the essential factors in causation, whilst in a third group the aneurysms owe their origin to the local action of infective emboli, and in a fourth are due to local non-granulomatous inflammatory changes in the wall of the artery.

Newton Pitt [16], in his third Goulstonian Lecture delivered in 1890, gave an account of 23 cases of cerebral aneurysms which had occurred amongst nearly 9,000 patients who had been examined *post-mortem* at Guy's Hospital between the years 1869 and 1888. His main conclusions were: (1) It is most exceptional to find a cerebral aneurysm which is not associated with fungating endocarditis. (2) The lodging of a septic embolus is the starting point of the changes which take place in the vessel. (3) The clot inflames and in most cases disappears, the vessel dilating from the inflammatory changes in its coats. (4) An aneurysm may form and rupture in three weeks. (5) The onset of the embolism may not be marked by any symptoms, and the rupture of the aneurysm may be the first indication that there is danger.

In 1893 Gowers [10], in his manual of diseases of the nervous system, gave a general account and review of the subject of intracranial aneurysm and discussed the causes, general pathology, symptomatology, course, diagnosis and prognosis. Amongst causes he discussed (1) primary degeneration of the arterial walls, (2) injury, (3) syphilitic disease, (4) embolism. "Primary degeneration is an occasional cause in the second half of life . . . atheroma seldom leads to simple dilatation." With regard to syphilis he stated that "an aneurysm has been frequently met with in cases of constitutional syphilis in young adults, in whom no other cause of aneurysm was discoverable." "Embolism is probably the most frequent cause in the first half of life. There are few cases during this period, without indications of syphilis or injury, in which there is not evidence of past or present heart disease." He gave a list of the numerical frequency in 154 cases obtained by combining the statistics of various writers and found that there had been on the middle cerebral 44 cases, basilar 41, internal carotid 23, anterior cerebral 14, posterior communicating 8, anterior communicating 8, vertebral 7, posterior cerebral 6, and on the inferior cerebellar 3.

Von Hofmann [25] in 1894 published an account of the pathological changes and clinical manifestations which he had observed in 78 new cases of intracranial aneurysm. The order of frequency of affection of the various arteries in his cases was: (1) middle cerebral, (2) internal carotid, (3) anterior communicating, (4) basilar, (5) vertebral, (6) anterior cerebral. He found that the posterior communicating and posterior cerebral arteries had been only seldom affected, and did not find a single instance of a case of aneurysm of the cerebellar arteries. He observed that the favourite sites are (a) the bifurcations of the larger

vessels, and (b) the points of division of their branches. He discussed the relation of intracranial aneurysms to atheroma, syphilitic arteritis, embolism, and trauma, and concluded that neither atheroma, syphilis nor trauma are common direct causes of the formation of aneurysms; atheroma and a heightened-blood pressure are important factors in the causation of rupture, and the syphilitic endarteritis of Heubner may exceptionally give rise to aneurysmal formation. Of his cases 70 per cent. were females, and 30 per cent. were males. Fifty-eight were patients brought up to hospital by the police; 8 of these were found dead, 3 in the w.c., 2 in bed, 1 in a cellar, and 1 in a shop, and 8 others died before admission to hospital. He laid great stress upon the importance of looking for intracranial aneurysms in cases suspected of a violent death.

In 1901 Simmonds [23], following up the work of Ponfick and Eppinger, described the minute pathological and bacteriological features which he had found in seven cases of intracranial aneurysm due to infective emboli. All his cases were examples of progressive, malignant endocarditis where the septicæmia was of coccal origin; they occurred in children or young adults who at autopsy showed vegetations on the valvular endocardium and single aneurysms on the cerebral arteries, but no other abnormalities in the cardio-vascular system or changes in the kidneys.

Beadles [2] in 1907, in his Lecture before the Royal College of Surgeons, discussed the symptomatology of aneurysms of the larger cerebral arteries, basing his remarks on the notes of 555 cases of aneurysms of the cerebral arteries found after death, accounts of 441 of which had appeared in the literature and 114 were fresh material. At the end of the lecture he remarks that "notwithstanding the remarks made by some who have specially written on intracranial aneurysms and the writers of some of our leading text-books, the conclusion that I have been forced to draw from a careful study of a large series of cases is, therefore, that it is quite impossible to diagnose an aneurysm of any one of the cerebral arteries except in the most unusual circumstances. Only two or three have ever been diagnosed during life." "The apoplectic symptoms following rupture of an aneurysmal sac are indistinguishable from those of cerebral or meningeal haemorrhage from other causes. If there is any one sign to which special attention might be drawn, it is the occasional intermittent character of the symptoms. But these form a very small proportion even of the cases where tumour symptoms were present, and, after all, it is not very

uncommon for symptoms to show variations in intensity, or even a temporary disappearance, in some other form of tumour and in other cerebral diseases."

The relative importance which, in recent years, authorities have placed upon the different factors which are known to be concerned with the formation of intracranial aneurysms have varied greatly. Thus in a clinical lecture delivered in 1908 John Rose Bradford [4] said: "The formation of a cerebral aneurysm is due to local disease of a vessel; it is a manifestation often of a patchy disease and not of a general arterial disease. . . . The local disease is, in a very large proportion of cases, atheroma, and is atheroma of no doubt syphilitic origin, and we think that in cases of cerebral aneurysms their causation by syphilis is, if anything, a more definite characteristic than it is even in such aneurysms as those of the aorta. In all the cases from which these specimens were removed where a history was obtainable there was no difficulty in tracing the syphilitic infection. . . . So much is that the case that you might make a more general statement and say that in aneurysms involving the larger vessels in young persons the probability of the condition being syphilitic is great. But bear in mind that in the case of the cerebral vessels, great as is the part played by syphilis, there is another factor which does not play any considerable part in the production of aneurysms elsewhere, that is the factor of embolism." Bruns [6], on the other hand, writing in the same year on the pathology of cerebral aneurysms, said that "in the majority of cases the aneurysms are due to arteriosclerosis and atheroma of the intima" and only admitted of syphilis as a cause of these underlying conditions.

Wichern [27] in two recent papers has reported the clinical histories and *post-mortem* findings in 22 new cases and has contributed to the better understanding of the clinical manifestations and to the diagnosis of the condition in patients suffering from aneurysms of the intracranial arteries. According to his analysis of his own cases, 5 were certainly and 6 probably of congenital origin, 5 were embolic, 2 were syphilitic, 2 occurred in patients suffering from general arteriosclerosis, and 2 were "atheromatous," but as far as can be judged from his paper Wichern uses the term "atheroma" to cover changes both in the intima and in the media of the arterial wall. In every one of Wichern's cases the aneurysmal sac had ruptured and in 15 of the 22 clinical or *post-mortem* evidence of previous leakage or rupture, on one or more occasions, was obtained. He insists that aneurysms of cerebral arteries are not rare

and that the infrequency of diagnosis in patients is due to the fact that the possibility of cerebral aneurysm as a diagnosis is rarely considered.

Chapter II.—The Pathology of Intracranial Aneurysms.

Turnbull [24] divides the chief abnormalities which affect the arteries into: (1) hypertrophies; (2) degenerations; (3) infiltrations; (4) inflammations, and (5) congenital malformations. He confines the term *inflammation* to reactions which are essentially active, and uses the term *degeneration* to distinguish those processes in which degeneration and necrosis are the primary and essential lesions. Only inflammations, degenerations and congenital structural malformations bear any causative relationship to the formation of intracranial aneurysms.

Atheroma is a degeneration which affects and is almost confined to the intima; it is found in both elastic and muscular arteries but is commoner in the large elastic; it is the result and not the cause of the increased blood-pressure which usually accompanies its presence; there is no evidence to be obtained in the *post-mortem* room to show that syphilis induces atheroma.

He finds that medial degenerations have the same cause as atheroma and may assume the forms of (*a*) fatty degeneration; (*b*) calcification associated with fatty degeneration; (*c*) mucous degeneration; (*d*) medial fibrosis.

In inflammations the arteries may be infected from their intima, either by micro-organisms settling upon the surface or by the arrest of infective emboli within their lumen. They may also be infected by micro-organisms reaching the media or adventitia by the vasa vasorum, or by direct inward spread of inflammation from the surrounding tissues. Inflammations of arteries may be divided into the *specific granulomatous inflammations* caused by syphilis, tuberculosis, rheumatism, &c., and *non-granulomatous* where the reaction is caused by the common pyogenic organisms. In his paper he separates for convenience of description the inflammations as they occur in the large elastic arteries, and in the muscular and small elastic arteries, and shows that acquired syphilis is far the commonest cause of aortitis, and is also the commonest cause of inflammation of the other large elastic arteries near the aorta, and, moreover, that syphilitic aortitis is the lesion of acquired syphilis which is found most frequently in

the *post-mortem* room of a General Hospital. In the small muscular and elastic arteries, the group to which the cerebral arteries belong, syphilis induces a relatively chronic arteritis, usually spoken of as endarteritis obliterans, in which changes in the intima are conspicuous and produce almost complete occlusion of the lumen. In the cerebral arteries, especially those of the base, arteritis consequent upon syphilis causes the artery to appear to naked-eye examination fusiform, white and thick, to feel firm and solid, and on transverse section to show a cut surface of firm, grey tissue, in the centre of which a minute red lumen may be visible. Microscopically, endarteritis obliterans is characterized by the appearance of an intimal thickening internal to the elastic lamina and the boundary stripe. In the earliest stages round cells are seen between the stripe and the endothelium. Spindle fibroblasts are found later and give rise to the formation of a definite granulation tissue. The granulation tissue ultimately becomes fibrotic and delicate elastic fibrils make their appearance. The elastic lamella and stripe remain unaltered throughout the process; this enables the condition to be recognized easily in sections stained by a specific method for demonstrating elastic tissue and to be distinguished readily from medial and intimal degeneration. The inflammation of the media and adventitia which accompanies the endarteritis conforms in type to the inflammation caused in other tissues by the particular micro-organism.

Turnbull discounts the use of the term *arterio-sclerosis* because it has been applied indiscriminately to hypertrophied arteries, to arteries showing atheromatous or (and) medial degeneration, to arteries infiltrated with amyloid, and to arteries reacting by inflammation. The term means merely "arterial hardening" and does not describe the pathological processes at work.

The term aneurysm (*ἀνευρύνω*, widen or dilate) is used to describe any dilatation of an artery. In this paper, as in Turnbull's, however, we shall deal only with focal, true, fusiform or saccular aneurysms of a size large enough to be readily visible to the naked eye. *True aneurysms* are dilatations which are bounded by the coats of an artery. *False aneurysms*, on the other hand, are cavities which communicate with the lumen of an artery but are bounded by tissues external to the wall of the artery. True aneurysms frequently rupture and give rise secondarily to false aneurysms; the resulting structure is spoken of as a *mixed aneurysm*. In this paper, all false aneurysms in which there is evidence of a primary dilatation, that is to say, all mixed aneurysms,

will be referred to as true. The term "direct rupture" will be employed to denote a rupture which has not been preceded by aneurysm. False aneurysms will, therefore, be included among "direct ruptures."

According to Turnbull, true aneurysms thus described may be divided pathologically into two main groups: (1) *congenital*; (2) *acquired*. In the formation of acquired aneurysms generally, trauma, peptic digestion, inflammations, both granulomatous and non-granulomatous, degenerations, loss of mechanical support and heightened blood-pressure are the only known factors at work. Turnbull has not been able to find, either in the *post-mortem* room or from a study of the literature, any unequivocal evidence that aneurysms of the cerebral arteries are ever due to trauma; digestion by peptic ferments introduced from without and loss of mechanical support also cannot be assigned as important factors in the formation of intracranial aneurysms. The only factors which remain to be considered, therefore, are: (1) congenital; (2) inflammatory, and (3) degenerative.

In the series of cases which Turnbull and I have studied and which form the basis of this paper, no single example of granulomatous inflammation giving rise to a cerebral aneurysm occurred. Such cases, however, in the literature are not rare. They have been found most commonly on the vertebral and basilar system of the basal arteries and descriptions of their morbid characteristics have been given by many authors; Rose Bradford [3], for example, in 1894, showed a good example which occurred on the basilar artery in a patient who at autopsy also showed multiple gummata of the liver. Wichern [28] described two cases (Cases 19 and 20) of ruptured aneurysm of the basilar artery accompanied by diffuse syphilitic endarteritis obliterans of the cerebral vessels; the first was found in a widow of 56, who at autopsy also showed periostitis of the tibia and syphilitic mesaortitis, and the second in a woman of 51.

Unfortunately, as in the majority of my own cases, Wichern's patients were observed some years ago and before the recent development and application of the Wassermann technique, and so far I have not been able to find any more recently described case where the Wassermann reaction was tested both in the serum and in the cerebrospinal fluid *ante mortem* and where at autopsy an aneurysm of one of the cerebral vessels was discovered. In 1913, Langbein [14] published a description of a single case in which a positive Wassermann reaction in both the serum and in the cerebrospinal fluid was found in a man aged 40, which clinically simulated and which he himself

recorded as one of recurrent leakages on the part of a cerebral aneurysm; but the patient recovered and the nature of the meningitic affection and the cause of the recurring apoplectic attacks remain extremely doubtful, as no autopsy has as yet been performed.

The relatively high incidence of syphilitic aneurysm of the basilar artery is probably due to the fact that this artery is a large one and that in the smaller cerebral arteries syphilitic inflammation usually leads to such a severe degree of intimal hypertrophy that the lumen of the artery is blocked and thus prevents aneurysmal dilatation. When an extremely intense arteritis, whether granulomatous or non-granulomatous, affects the cerebral vessels, direct rupture or the formation of a false aneurysm may take place.

Aneurysms of the muscular and small elastic arteries frequently result from infection by bacteria which cause acute inflammations. The arteries are most commonly infected by impaction of emboli in their lumina. Aneurysms due to infective embolism are found most frequently in the cerebral arteries; the superior mesenteric artery is the second site of election. In our records they have occurred most frequently in cases of progressive malignant endocarditis. No aneurysms of cerebral vessels were caused by infection from without.

Saccular aneurysms due to medial degeneration, independent of inflammation, are found much more commonly on the cerebral arteries than on any other muscular arteries. In none of the cases of cerebral aneurysm which we have attributed to this class was there evidence at necropsy of active or healed endocarditis. In some there was evidence of cardio-vascular hypertrophy, accompanied by a considerable degree of general arterial degeneration, and in such cases, as Turnbull remarks, there would appear to be an adequate explanation of the formation of the aneurysms. In many of the cases, however, there was no evidence of excessive blood-pressure, either at autopsy or from clinical observations, and the general arterial degeneration and the degeneration of the cerebral arteries were no greater than in the average subject of the same age. In this respect many of the cases of aneurysm contrast sharply with the cases of direct rupture of central cerebral arteries. Direct rupture is usually associated with evidence of much more excessive blood-pressure (hyperpiesia) and arterial degeneration. There appears, therefore, to be an additional factor in the formation of cerebral aneurysms. This factor is, probably, (as Eppinger [9] first suggested) an inherent, inborn weakness due to a congenital abnormality in the structure of the arteries at their

points of junction and bifurcation. This conclusion is strengthened by the finding in the *post-mortem* room of the London Hospital in the year following the cases analysed in this paper of an aneurysm at the junction of the left anterior cerebral with the anterior communicating artery, in a child aged 1 year and 7 months, who died of broncho-pneumonia complicating acute gastro-enteritis.

The arteries of the brain all receive their blood from the internal carotid and vertebral arteries, whose anastomoses at the base of the brain form the arterial circle of Willis. On the ventral surface of the fore end of the medulla oblongata the two vertebral arteries unite to form the unpaired basilar artery; this artery courses over the pons and divides anteriorly into the two posterior cerebral arteries which run out almost at right angles to the parent vessel. The internal carotid arteries immediately after reaching the cranial cavity send back the posterior communicating arteries which unite the posterior cerebrals with the carotid system. On the side of the optic tract the internal carotid arteries give off the anterior cerebral arteries, which supply the frontal lobes and corpus callosum of the brain, and continue as the the middle cerebral arteries. The two anterior cerebral arteries are united by the unpaired anterior communicating artery.

The circle of Willis is thus formed by:—

basilar artery { posterior cerebral—posterior communicating—
{ posterior cerebral—posterior communicating—

—carotid—anterior cerebral }
—carotid—anterior cerebral } anterior communicating.

Although this is the "normal" arrangement, variations are extremely common. Amongst 200 successive observations on the conformation of the circle of Willis in dissecting-room subjects Windle [29] found that the number of arteries and their arrangement was "normal" in 119 (59·5 per cent.); the circle was symmetrical in 76, and symmetrical but for some inequality in size of the posterior communicating arteries in 43 others. In 159 the anterior communicating artery was normal; in 14 cases two anterior communicating arteries were present, in 1 case three were found, in 6 cases the artery was incompletely duplicated, in 6 cases the anterior cerebral arteries were united at their origin, in 2 cases both an union of the anterior cerebral arteries at their origin and also an ordinary anterior communicating artery were found, and in 9 cases a mesial artery arose from the anterior communicating artery. In 181 the anterior cerebral

arteries were normal; in 9 cases two anterior cerebral arteries were discovered with in addition a mesial artery arising from the anterior communicating artery; in 8 cases the anterior cerebrals were united for a distance and in 3 cases only a single mesial anterior cerebral artery was found. In 173 the posterior cerebral arteries and in 175 the posterior communicating arteries were normal. In size the posterior communicating arteries were often unequal, the right was larger than the left in 28 cases, and the left larger than the right in 15; both were small in 7 cases and both were absent in 3 cases; in 9 the right was absent and in 13 no left posterior communicating artery could be found. Wyrubow [30] amongst 112 successive examinations of the conformation of the circle of Willis in patients who had died from mental disease found that 22·3 per cent. showed abnormalities of the anterior cerebral arteries, and Parnisetti [19] at the autopsies on 87 criminals found only 51 (58·62 per cent.) with normal arrangements of the circle of Willis. Rothmann [22] states that in apes and anthropoids the anterior communicating artery is usually absent and the circle of Willis is thus not completed. Hofmann [13] found that the circle is usually incomplete anteriorly in frogs, salamanders and birds, and showed that amongst the various sub-groups of the mammalia the arrangements vary: zoologically closely related mammalia often show important differences in the arrangement of the basal cerebral arteries. Thus amongst the Ungulata, in the pig the circle is usually completed anteriorly; in the cow, on the other hand, this is not so. Amongst the Anthropoidea, in the gorilla no anterior communicating artery is found; in man this artery is normally present.

From these observations we see that morphological variations of a gross character are extremely common in the arteries forming the circle of Willis in all classes of the Vertebrata, and microscopical investigations have shown that minor changes in the elastic layer of the media of the arteries are also common in the cerebral arteries of patients who show no gross saccular aneurysmal dilatations. Most of the variations, whether major or minor, are found at the points of junction or of bifurcation of the vessels, and it is at these points that we find aneurysms visible to the naked eye of supposedly congenital origin.

In no case in this series of cases could any relation to trauma as a possible cause of the formation of intracranial aneurysm be traced.

Occurrence.—During the years 1907 to 1913 at the Pathological Institute of the London Hospital 7,924 *post-mortem* examinations of the body were made, in 5,432 of which an examination of the head was permitted. In these cases the following aneurysms were discovered:—

True Aneurysms of Aorta—	
Due to syphilitic inflammation	175
,, tuberculous inflammation	1
,, non-granulomatous inflammations	4
,, atheromatous degeneration	4
	184
Dissecting Aneurysms of Aorta—	
Due to non-granulomatous inflammation	5
,, developmental deficiency	2
	7
True Aneurysms of Other Large Elastic Arteries—	
Due to syphilitic inflammation	43
True Aneurysms of Muscular and Small Elastic Arteries (excluding the Cerebral Arteries)—	
Due to syphilitic inflammation	6
,, tuberculous inflammation (probably not all reported, most occur with pulmonary vomicæ)	10
,, infective embolism	18
,, spread of inflammation from without	3
,, medial degeneration	7
	44
True Aneurysms of Cerebral Arteries—	
Due to infective embolism	15
,, medial degeneration	36
	51

From this table we see that aneurysms of the aorta occurred in 191 cases or in 2·4 per cent. of examinations of the body, that 51 aneurysms of the cerebral arteries occurred in 44 cases or in 0·80 per cent. of examinations of the head, and that 44 aneurysms of the other large elastic arteries also occurred, i.e. one in 0·54 per cent. of examinations of the body. These figures contrast strongly with those from Crisp which Osler [17] quotes in his "System of Medicine," where amongst 501 aneurysms, 184 occurred in the aorta, 137 in the popliteal artery and only 7 intracranial aneurysms were discovered.

The commonest cause of all aneurysms is syphilitic inflammation of the wall of an artery. Ninety-six per cent. (96·2 per cent.) of the aneurysms of the aorta, 100 per cent. of the aneurysms of the other large elastic arteries, but only 6·25 per cent. of the aneurysms of the muscular and small elastic arteries, to which class the arteries of the circle of Willis and the cerebral arteries generally belong, were consequent on the local weakening of the arterial wall due to the inflammatory reaction consequent on the activity of the *Spirochæta pallida*, and in no case in this series of necropsies was an intracranial aneurysm due to this cause found; at the same time the low rate of incidence of aneurysms on the vertebral system of arteries in this

series of cases, compared with those recorded by others, is extremely noteworthy.

In this series of examinations of the brain 31 cases showed thirty-six aneurysms of the cerebral arteries due to non-inflammatory medial change, and 13 cases showed fifteen cerebral aneurysms caused by infective embolism. Aneurysms due to non-inflammatory medial change occurred therefore in 0·55 per cent. of examinations of the brain, and aneurysms due to inflammatory changes in the cerebral arteries associated with infective embolism in 0·24 per cent.

Of these aneurysms on the cerebral arteries, death had been caused by rupture in 25 cases where the aneurysm was due to non-inflammatory changes, and in 10 cases where the aneurysm had resulted from infective embolism, or together in 4·42 per mille of the autopsies at the London Hospital during the years 1907 to 1913. During the same period intracranial hæmorrhage due to the direct rupture of central cerebral arteries consequent on medial degeneration was found in 72 cases or in 9·08 per mille of the autopsies, giving the relative frequency of cerebral hæmorrhages due to direct rupture compared with those following true aneurysmal formation of 35 to 72, or 1 to 2·05.

The following is a list of the sites of the cerebral aneurysms classified according to their pathological causes:—

	Embolic		Medial degeneration
Left intracranial carotid	1	..	1
Origin of cortical branch from left carotid	0	..	1
Junction right carotid with posterior communicans ..	0	..	1
Junction left carotid with posterior communicans	0	..	2
Junction right carotid with middle cerebral	0	..	2
Right middle cerebral near base of brain	1	..	8
Left middle cerebral near base of brain	1	..	2
Cortical branches of right middle cerebral	1	..	2
Central branches of right middle cerebral	0	..	1
Cortical branches of left middle cerebral	3	..	0
Central branches of left middle cerebral	1	..	0
Junction of right anterior cerebral with anterior communicans	0	..	2
Junction of left anterior cerebral with anterior communicans	0	..	7
Right anterior cerebral	2	..	2
Left anterior cerebral	1	..	1
Left anterior communicans	0	..	1
Junction of right posterior cerebral and posterior communicans	0	..	1
Bifurcation of basilar	1	..	0
Left posterior cerebellar branch of basilar	0	..	1
Left anterior inferior cerebellar	0	..	1
	15	..	36

Multiple cerebral aneurysms due to medial change were found in four of the thirty-one cases:—

Case 3 showed two aneurysms on central branches of the right middle cerebral artery, one of which had ruptured.

Case 4 exhibited two intracranial aneurysms, one of which at the junction of the left anterior cerebral and the anterior communicating arteries had ruptured, and the other of which at the junction of the right carotid and posterior communicating arteries was unruptured.

Case 11 showed two aneurysms, neither of which had ruptured; one of these was found on the right middle cerebral artery near the base of the brain, and the other at the junction of the left anterior cerebral and anterior communicating arteries.

Whilst Case 26 showed three aneurysms; one occurred at the junction of the left intracranial carotid and posterior communicating arteries and was ruptured, and the other two were found on the right middle cerebral artery and had not ruptured. Thus amongst 31 cases showing intracranial aneurysms, due to medial change, 4 cases exhibited nine aneurysms, and 31 cases thirty-six aneurysms.

Fifteen saccular aneurysms due to medial degeneration occurred at the actual point of junction of the arteries of the circle of Willis (Cases 4 (three examples), 5, 6, 8, 10, 11, 16, 17, 23, 24, 26, 27, 30). In addition, six were found close to such junctions and ten arose from the point of origin of branches. In two cases no note was made upon the relation to points of junction or bifurcation.

In one case two minute aneurysms were situated upon central cerebral branches. Of the remaining 34 due to medial degeneration, all except one sprang from cerebral arteries of the first order. Only three were connected with the posterior part of the circle of Willis.

The various sites of the intracranial aneurysms caused by infective embolism which occurred during the years 1907 to 1913 are given in the table on next page.

In this table are shown nineteen aneurysms of the muscular and small elastic arteries which occurred in 13 cases. Amongst these cases were found three aneurysms on visceral arteries and one on the profunda femoris artery. During the same period in cases where an examination of the head was permitted and no cerebral aneurysms were found, there occurred two aneurysms on the superior mesenteric artery, two on the splenic artery and one on the left brachial artery, giving the proportion of aneurysms due to infective embolism amongst 5,324 examinations of fifteen cerebral to nine on other arteries. In the 13 cases showing at autopsy cerebral aneurysms, ten of the cerebral

Number of cases	Right middle cerebral	Left middle cerebral	Branch on vertex right middle cerebral	Branch on vertex left middle cerebral	Right inferior cerebral	Left anterior cerebral	Left intracranial carotid	Bifurcation of basilar	Superior cerebellar	Ocular axis	Left retinal artery	Left profunda femoris	—
1	R												I, IV, VIII, X.
1	4R												II.
1	R				1R	1							VI.
1			1R										VII.
1		1R		1									III.
1		1R					1			1R	1R	1	V.
1								1					IX.
1		1R							1				XI.
1		1R			1R								XII.
13	4	4	1	1	2	1	1	1	1	1	1	1	
	3R	3R	1R		2R								

aneurysms and two of the other aneurysms had ruptured and given rise to considerable hæmorrhage. *Post-mortem* evidence of progressive malignant endocarditis was found in 10 of the cases; in 1 case a pyæmia subsequent to a retropharyngeal abscess, and in 1 case a pyæmia consequent on puerperal sepsis were found, and in 1 case the primary seat of the infection was not ascertained.

Multiple aneurysms due to emboli in cases of general septicæmia occurred in two cases.

Case V showed a ruptured aneurysm on a perforating branch of the left middle cerebral artery and an unruptured aneurysm on a cortical branch of the same artery: whilst Case XIII showed two ruptured cerebral aneurysms, one on a cortical branch of the left middle cerebral artery and a second on a cortical branch of the right anterior cerebral artery.

In 3 cases aneurysms on arteries in other parts of the body were also found.

Case V showed a ruptured aneurysm on the left profunda femoris artery; Case IX showed a ruptured aneurysm on the cœliac axis artery and an unruptured aneurysmal dilatation of the left renal artery; and Case XII an unruptured aneurysm on the superior mesenteric artery. Thus, of 13 cases 2 showed multiple intracranial aneurysms and 3 showed aneurysms of vessels in other parts of the body.

In this series of cases 17 intracranial aneurysms due to medial changes occurred on the left side, 19 on the right, whilst of those caused by infective embolism 7 were found on the left side and 7 on the right and 1 occurred at the bifurcation of the mesial basilar artery. Thus 24 occurred on the left half of the body and 26 on the right.

Size—

(a) Of Cerebral Aneurysms due to Medial Degeneration.

Smaller than a pin's head	Case 11	1
Size of a pin's head	Cases 10, 11, 21, 28	4
Size of half-pea	Case 3	1
Size of a grape-stone	Case 26 (two examples)	2
Larger than a grape-stone	Case 26	1
Size of a pea	Cases 1, 3, 4, 4, 5, 6, 7, 12, 16, 20, 27, 29	12
Size of a cherry-stone	Cases 13, 15, 25	3
Size of a currant grape	Case 23	1
Size of a haricot-bean	Case 31	1
2 mm. in diameter	Case 2	1
3 mm. in diameter	Case 19	1
6 mm. in diameter	Cases 8, 17, 18	3
14 mm. in diameter	Case 24	1
15 mm. by 10 mm.	Case 30	1
30 mm. in diameter	Case 14	1
No notes on size	Cases 9, 22	2
		36

(b) Of Cerebral Aneurysms due to Infective Embolism.

Size of a pea	Cases II, V, VI	3
Size of a cherry-stone	Cases IV, VIII, X	3
Size of two peas	Case I	1
Size of three peas	Case III	1
Size of a cherry	Case VII	1
"Dilatation of vessel"	Cases V, IX, XI	3
25 mm. in diameter	Case XII	1
No notes on size	Case XIII (two examples)	2
		15

Sex.—Cerebral aneurysms due to medial degeneration were found in 18 males and in 13 females, and due to infective emboli in 8 males and 5 females, giving a proportion in the whole series of cases of 26 males to 18 females.

Age.—If we divide up our cases of saccular aneurysms of cerebral arteries due to changes in the media according to the degree of cardio-vascular hypertrophy we find that:—

In two patients, aged 51 and 48, there was *very great* cardio-vascular hypertrophy.

In four patients, whose ages varied from 60 to 44, and averaged 50, there was *great* cardio-vascular hypertrophy.

In three patients, whose ages were 86, 75 and 50, there was *considerable* cardio-vascular hypertrophy.

In three patients, aged 55, 53 and 46, there was *moderate* cardio-vascular hypertrophy.

In three patients, aged 72, 67 and 60, there was *slight* cardio-vascular hypertrophy.

In fifteen patients, whose ages varied from 53 to 19 and averaged 38, there was *no* cardio-vascular hypertrophy.

And these cases were distributed between	50 and 54	2
,, ,, ,, ,,	45 ,, 49	3
,, ,, ,, ,,	40 ,, 44	4
,, ,, ,, ,,	35 ,, 39	2
,, ,, ,, ,,	30 ,, 34	0
,, ,, ,, ,,	25 ,, 29	1
,, ,, ,, ,,	20 ,, 24	2
,, ,, ,, ,,	15 ,, 19	1

and in one case only an examination of the head was permitted.

A study of the incidence, therefore, in these thirty-one cases shows:—

That between the ages of	85 and 89 there occurred	1 case
,, ,,	80 ,, 84 ,,	0 ,,
,, ,,	75 ,, 79 ,,	1 ,,
,, ,,	70 ,, 74 ,,	1 ,,
,, ,,	65 ,, 69 ,,	1 ,,
,, ,,	60 ,, 64 ,,	2 cases
,, ,,	55 ,, 59 ,,	0 ,,
,, ,,	50 ,, 54 ,,	6 ,,
,, ,,	45 ,, 49 ,,	7 ,,
,, ,,	40 ,, 44 ,,	6 ,,
,, ,,	35 ,, 39 ,,	2 ,,
,, ,,	30 ,, 34 ,,	0 ,,
,, ,,	25 ,, 29 ,,	1 case
,, ,,	20 ,, 24 ,,	2 cases
,, ,,	15 ,, 19 ,,	1 case
		31

The age-incidence of cerebral aneurysms of embolic origin is determined by that of chronic septicæmic and pyæmic conditions. In this series of cases

Between the ages of	5	and	9	there occurred	..	..	1	case
,, ,,	10	,,	14	,,	..	..	0	,,
,, ,,	15	,,	19	,,	..	..	4	cases
,, ,,	20	,,	24	,,	..	..	3	,,
,, ,,	25	,,	29	,,	..	..	0	,,
,, ,,	30	,,	34	,,	..	..	1	case
,, ,,	35	,,	39	,,	..	..	2	cases
,, ,,	40	,,	44	,,	..	..	2	,,
							13	

Chapter III.—The Clinical Manifestations associated with Intracranial Aneurysms of Non-Embolic Origin.

The course of illness and the actual manifestations of departure from healthy states which occur in patients who at autopsy exhibit aneurysmal dilatation *with rupture* of the larger cerebral arteries form a fairly definite nosological group. Nevertheless these deviations from the normal so closely resemble those seen in morbid intracranial conditions owning other causes that Gull's statement written in 1859 still holds true "that although we may from the circumstances sometimes suspect the presence of an intracranial aneurysm, we have at the best no symptoms upon which to ground more than a probable diagnosis.

In nervous tissue, as contrasted with the other tissues of the body, function is more strictly localized. The interferences with function which result from the appearance of morbid changes in the vessels supplying the central nervous system depend upon the particular portions of the nervous axis which are affected secondarily. The finding on a given date of no single set of clinical manifestations of itself can enable us to pronounce a definite opinion upon the nature of the underlying pathological processes; tumour, syphilitic, and other forms of meningitis, aneurysm, &c., occurring at the same point, give rise to identical interferences, but for each the course of the disease is more or less characteristic, and if the possibility of aneurysm as a diagnosis were more frequently considered a larger number of cases would be recognized during life. In a considerable proportion of the cases which are included in this series a shrewd guess as to the causative factor was made before autopsy, and in a still larger number such a guess ought to have been hazarded. In the *post-mortem* room an aneurysm of one of the cerebral

arteries is a relatively common finding, and yet in the past when considering at the bedside the causative possibilities in a given patient cerebral aneurysms have rarely been suggested.

In 80·7 per cent. of the cases (25 out of 31) of non-embolic aneurysms due to changes in the media included in this series, signs attributable to cerebral hæmorrhage or to a local affection of the meninges consequent upon the *rupture* of the sac occurred, whilst in 16·1 per cent. (5 out of 31) an unruptured cerebral aneurysm, which, so far as is known, had not led during life to any definite manifestations, was found, and in 3·2 per cent. (1 case out of 31) in the history given by the patient on his admission to hospital was an account of a seizure and of an "influenzal" attack followed by much frontal headache and deafness of the right ear, which might or might not have been due to the development of an aneurysm at the junction of the right carotid and middle cerebral arteries.

Cerebral aneurysms which do *not* rupture rarely give rise to symptoms.

Death occurred in Case 7 from broncho-pneumonia consequent upon septic pharyngitis with abscess formation in the neck in a man aged 86, who five weeks previously awakened one morning complaining of an inability to utter words. At the autopsy atheroma of most of the cerebral arteries and unruptured aneurysm, of the size of a pea, was found on the right middle cerebral artery near the base of the brain. In Case 8 the patient, a man aged 75, died forty hours after a suprapubic prostatectomy and an unsuspected and unruptured aneurysm, 6 mm. in diameter, was found at the junction of the left anterior cerebral and anterior communicating arteries. Case 11, a fish-curer, aged 53, for two and a half years before death suffered from hæmaturia and renal insufficiency associated with congenital cystic kidneys. He died "uræmic," and at autopsy two small unruptured aneurysms were found, one at the junction of the left anterior cerebral and anterior communicating arteries, of the size of a pin's head, and the other, rather smaller, on the right middle cerebral artery near the base of the brain.

Case 19 was that of a married woman aged 48; for five years she had suffered from symptoms attributable to cholelithiasis, and eighteen days before death was admitted to hospital with an acute surgical condition of the abdomen. Laparotomy was immediately performed; a tumour of the gall-bladder was removed and a subphrenic abscess drained. After the operation her general condition grew worse, hæmatemesis from acute ulceration of the stomach set in and she died of

internal hæmorrhage. At the autopsy an unruptured aneurysm on the right middle cerebral artery, 3 mm. in diameter, was found near the base of the brain. Case 21 was admitted to hospital with symptoms of Korsakow's psychosis and alcoholic peripheral neuritis, from which she died. At autopsy cirrhosis of the liver, fibrosis of the pancreas and a certain degree of cirrhosis of the kidneys, and in addition an unruptured aneurysm, of the size of a pin's head, on the left middle cerebral artery about 1 in. from its origin, were found.

Case 20 is our only example of a patient in whom an unruptured aneurysm at the time of its development probably caused certain intracranial disturbances which came on acutely and then remained quiescent until the death of the patient from an intercurrent disease bearing no direct relation to the vascular system. This man, when aged 47, was admitted to hospital with signs of acute diffuse myelitis from which he died. He gave the history that six years before death he had suffered a slight seizure followed by general weakness, irritability and depression, and that about a year previously he had had an attack of "influenza" followed by deafness of the right ear and much frontal headache. Here an unruptured aneurysm, of the size of a large pea, was found at the junction of the right carotid and middle cerebral arteries, in addition to diffuse inflammation of the spinal cord.

Two of the patients included in this series *died before admission to hospital*. Of the previous history of Case 1 nothing is known. The patient, a man aged about 51, was found by the police lying dead in the street; a ruptured aneurysm of the size of a pea was found on the right middle cerebral artery near the base of the brain. The case was one of hyperpiesia; atheroma was present in the wall of the aneurysm, in most of the cerebral arteries and in most of the systemic arteries, and the heart showed left-sided hypertrophy. Case 28 was that of a clerk, aged 22, who had gained some local fame as a cyclist; after riding a mile at a public cycle-race meeting he "came over giddy," got off his machine, lay down on the side of the track, lost consciousness almost immediately and died within half an hour of the onset of the symptoms. At the autopsy a ruptured aneurysm on the right middle cerebral artery, 4 cm. from its origin and near the base of the brain, of the size of a pin's head, was discovered; in the rest of the cardiovascular system no gross changes were found.

Ten cases were admitted *after a first apoplectic seizure, and died without ever recovering consciousness*. Case 9, a man aged about 50, lived only one hour and a half after his admission; in this case the

diagnosis of "fractured base" was made owing to the history of a fall in the street and the finding on examination of a bruise on the occiput. At the *post-mortem* examination a ruptured aneurysm was found on the right anterior cerebral artery just beyond the origin of the anterior communicating artery: and in the great longitudinal fissure old altered blood-clot and staining of the meninges was present, suggesting that a partial rupture had occurred on some previous occasion.

Case 10 was that of a greengrocer, aged 55; he, too, was found lying in the street by the police and died in coma about an hour after admission. This patient showed left hemiplegia, unequal fixed pupils—the right larger than the left—hyperpiesia and enlargement of the left ventricle. At the autopsy a ruptured aneurysm of the size of a pin's head was found at the junction of the right anterior cerebral and anterior communicating arteries.

Case 24 died unconscious within a few hours after admission. The patient was a woman hawker, aged 40, who was found by the police lying stertorous and deeply cyanosed on the roadside. On admission, both pupils were widely dilated, and neither reacted to light, and both plantar reflexes gave an extensor response. Here the ruptured aneurysm, 1·4 cm. in diameter, was found at the junction of the right posterior cerebral and posterior communicating arteries.

Case 18 was that of a married woman, aged 49, who was said to have suffered for four months from nocturnal frequency of micturition, occasional attacks of headache, and a general nervous state of irritability attributed to the onset of the menopause. Eight days before death an extremely severe attack of headache leading to nausea and vomiting was complained of; the headache persisted for six days, and then the patient became delirious and lost consciousness. On admission ten hours before death she was deeply unconscious, and no local signs of interference with motion or with the functions of the cranial nerves were discovered. Two hours after admission she exhibited three generalized convulsions, developed hyperpyrexia (T. = 106° F.), and died. The aneurysm, which had ruptured, was found on the left anterior communicating artery, and was 6 mm. in diameter: there were few signs of cardio-vascular disease.

Case 27, a compositor, single, aged 28, had suffered for eighteen months from attacks of left-sided headache, accompanied during the last six months by severe nausea and giddiness, and usually some vomiting; sixteen hours before death he got up in the morning feeling

particularly well, and went to the bathroom, where he fainted and lost consciousness. Fourteen hours after this seizure he was admitted moribund, and within ninety minutes died with increasing coma and cyanosis. An aneurysm, of the size of a pea, which had ruptured, was found on the left intracranial carotid artery at the origin of a branch to the temporal pole. The rest of the cardio-vascular system showed few signs of disease.

Case 17 was that of a labourer, aged 51, who had been addicted to alcoholic excess, and for several winters subject to attacks of "winter-cough." About a month before death manifestations of cardiac failure appeared without any symptoms referable to the nervous system. Eighteen hours before death he was seized with a sudden intense headache, leading to vomiting and the appearance of a right-sided extensor plantar response; he lost consciousness, and died eighteen hours later. A ruptured aneurysm, 6 mm. in diameter, was found at the *post-mortem* examination on the left anterior communicating artery.

Case 13, an old woman, aged 72, who lived alone, was found lying unconscious in her kitchen; she died twenty-eight hours later. On admission neither pupil reacted to light, and both plantar reflexes gave an extensor response. An aneurysm, of the size of a cherry-stone, which had burst, was found on the left anterior cerebral artery just beyond the origin of the left anterior communicating artery. In this case there was moderate general arterial degeneration and moderate cardio-vascular hypertrophy.

A married woman, aged 48 (Case 2), was suddenly seized with apoplexy whilst in the street, and died unconscious thirty-six hours later. In this case widespread cardio-vascular changes were found at the autopsy, together with a ruptured aneurysm, 2 mm. in diameter, on the right middle cerebral artery near the base of the brain.

Case 5 was that of a man, aged 49, a painter by trade, who, whilst at work in the hospital garden, fell, and was found later lying unconscious. He was admitted immediately, but never recovered consciousness; he died on the third day after the seizure of respiratory failure. Lumbar puncture in this case showed the presence of much free blood in the cerebrospinal fluid, and the examination of the patient revealed general vascular disease, hyperpiesia, right facial paresis, and evidence of bilateral corticospinal interference. At the autopsy atheroma of most of the cerebral vessels, general arterial degeneration and hypertrophy of the left ventricle were found, and also a ruptured aneurysm of the size of a pea at the junction of the left anterior cerebral and anterior communicating arteries.

In Case 6 the patient, a married woman, aged 44, had suffered for several years from "ill-health, depression, and hysterical fits"; for these she took alcohol to excess. Twenty-four days before death she began to complain of severe pain in the head, acute diarrhœa, and much vomiting; this attack lasted three days. Then she improved somewhat, but remained drowsy, and talked irrationally. Fourteen days before death she was admitted to hospital in a drowsy, apathetic, lethargic state, passing urine and fæces under her. On examination, the calves were exquisitely tender to pressure, the knee-jerks and ankle-jerks were abolished, but the left plantar response was extensor; from time to time the patient showed attacks of irregular movements of the right upper extremity. In this state she remained for several days, then signs of broncho-pneumonia developed, and she died. The autopsy showed a ruptured aneurysm of the size of a pea at the junction of the left anterior cerebral and anterior communicating arteries, whilst both anterior cerebral arteries arose from the left internal carotid. All the cerebral and most of the systemic arteries showed medial and atheromatous changes, and the left ventricle was much enlarged.

The clinical history in this case, although the patient never regained complete consciousness after her first seizure, forms a connecting link with the more definite and more easily recognized group of cases in which multiple seizures at varying intervals and signs of localized interferences at the base of the brain precede the fatal issue.

When death in a case of ruptured cerebral aneurysm follows directly upon the first rupture of the sac, the differential diagnosis between intracerebral and intrameningeal hæmorrhage can only be made after a full consideration of all the attendant *localizing* symptoms and signs; and these often are insufficient to lead to the formation of a correct opinion. In 41·9 per cent. of cases (13 out of 31) of cerebral aneurysms of non-embolic origin in this series, however, an especial feature, which, so far as my experience goes, amongst all other intracranial conditions is characteristic of ruptures of this class of aneurysms, was also present, viz.: an account in the history of the patient's manifestations of a sudden exacerbation, with apoplectiform onset, of symptoms and signs followed by a period of varying, but often considerable, duration, in which no fresh developments appeared, followed later by a second and a third, and even by a fourth, seizure, in the last of which death from respiratory failure and medullary pressure occurred, and correlated with this the finding at autopsy of

evidence of leakages of different ages owing to partial ruptures of the aneurysmal sac.

Wichern [27 and 28], from a study of the literature of cerebral aneurysms in general, found that multiple seizures had occurred in 57 out of 183 cases (31·1 per cent.), figures which correspond well with the 13 out of 44 cases (29·6 per cent.) of all classes of cerebral aneurysms which, on the one hand, manifested multiple seizures, and, on the other, were examined in the *post-mortem* room of the London Hospital during the period under consideration.

A case of aneurysm at the junction of the left internal carotid and posterior communicating arteries of congenital origin which first give rise to symptoms at the age of 19. Three partial ruptures giving rise to apoplectiform attacks. Admitted to hospital showing complete paralysis of the left third cranial nerve, optic neuritis on left and proptosis on right, and left cerebellar posture of head. Cerebrospinal fluid blood stained. Death from respiratory failure twenty-four days after onset of manifestations.

Case 30. 31256 1911. Arthur F., aged 19, painter, single, was admitted to the London Hospital, under the care of Dr. W. J. Hadley, on June 8, and died on June 24, 1911.

Until June 1, 1911, the health of this patient had been uniformly good. On the afternoon of that day, whilst at work, he was suddenly seized with acute pain in the head "as if the head would burst" and "a feeling of stiffness all down the left side of the neck." The headache led to vomiting; the vomiting did not relieve the pain nor the feeling of stiffness. He lay down for about two hours, when the headache became less severe. He was then able to walk the quarter of a mile from his work to his home. On the next day he still suffered from severe headache over the left half of his forehead and down the left side of his neck. On June 3 he got up but did not go to work; on this day the headache, though still present, was less severe. On June 4, the pains in his head again became more severe and his appetite failed. On June 5 the headache was still severe and he experienced difficulty in opening his left eye. On the morning of June 6 he was totally unable to open his left eye. On June 7 and 8 the headache was constant and severe. On June 8 he first sought medical advice; he was seen as an out-patient by Dr. Lewis Smith and admitted to hospital.

The patient was the second of four children: his two brothers and sisters were healthy and his father and mother were alive and healthy. As a child and young man his health had been good. The only illness he remembered was an attack of scarlet fever at the age of 10 years. He denied all venereal infection and all exposure. Previous to the onset of the illness for which he was admitted he had never suffered from headache.

The patient was a well-developed healthy-looking young man of 19. He

weighed 6 st. 11½ lb. He was drowsy and lethargic and only spoke when addressed. Speech was unaffected. Before June, 1911, he had never suffered from fits or other forms of seizure. He complained of a severe headache confined to the left half of his head and neck, "a dull, constant, heavy feeling, varying a little in intensity from time to time." The left side of his head was tender on pressure: the tenderness extended down over the left mastoid region and was general. Vomiting had occurred on June 1.

With the right eye the patient could see distant objects with fair ease, but with this eye he was unable to read the newspaper. Vision with the left eye was unimpaired. The arteries of the right fundus appeared normal, but the veins were engorged, whilst the disc appeared healthy; on this side there was some œdema of the lower quadrants of the fundus. On admission, the left fundus was not clearly seen, but in it no gross change was detected. Hearing, smell and taste were unaffected.

The left upper eyelid could not be raised, and of the muscles moving the left eyeball only the external rectus was not paralysed. The right eyeball was prominent but its movements were unimpaired. The right pupil was of moderate size and reacted readily to light and on accommodation. The left pupil was widely dilated and did not react either to light or to attempts at accommodation. The action of the muscles moving the jaws was normal. The right side of the face was flat and the nasolabial fold was more prominent on the left side than on the right, but in voluntary and emotional movements the face moved naturally. The movements of the palate and larynx were unimpaired. The tongue could be protruded straight and held steadily.

He walked naturally: gait was unaffected and Romberg's sign was not obtained. Whilst lying in bed he habitually held his head flexed towards the left with the chin pointing to the right. The muscles of the left side of the neck were in constant spasm. The grasps were powerful and co-ordination of the hands and legs was unimpaired. No anæsthesia or alteration in sensibility could be discovered to testing with the prick of a pin, cotton wool, the hot and cold tubes and for postures and passive movements.

The knee-jerks were readily obtained and about equal on the two sides. Ankle-clonus was not present and the arm-jerks were normal. The abdominal reflexes were readily elicited and both plantar reflexes gave a flexor response. The sphincters were under control.

The apex-beat of the heart was felt in the fifth space 2¾ in. from the mid-sternal line. The area of cardiac dulness was not increased. The heart sounds were pure. The rate of the pulse varied from 76 to 90 per minute and that of respiration from 20 to 24 per minute. No abnormal signs were found over the lungs. The temperature on admission was 100° F.; throughout the patient's stay in hospital the temperature oscillated in an irregular manner between 97° and 100° F. No abnormal signs were discovered in the abdomen; the liver and spleen could not be felt. The quantity of urine passed in twenty-four hours varied from 50 to 80 oz., the specific gravity from 1014 to 1028; neither albumen nor sugar was ever found. There was no glandular enlargement.

During the whole period of observation headache was a constant feature and was not affected by the administration of drugs. The pains in the left cervical region gradually spread down the spine as far as the lumbar region, but this spread was not accompanied by any exacerbation in the headache. On June 15, the prominence of the right eye, noted on admission, became more definite and was now associated with lateral nystagmus both to the right and to the left and with coarse hippus of the pupil of the right eye: on this day, too, Kernig's sign was readily elicited on both sides.

On June 15, and again on June 18, lumbar puncture was performed; on each occasion blood-stained fluid escaped at considerable pressure, and it was observed that the longer the cerebrospinal fluid ran the more blood the specimen obtained contained.

On the morning of June 24, five hours before death, the patient's condition was closely similar to that seen on admission. He was, however, more irritable and mentally more dull than he had been on the previous four or five days. The neck muscles on both sides were rigid and the patient deeply resented all passive movements of his neck. The grasps were feeble. All the limb muscles were hypotonic but there was no absolute paralysis. The knee- and ankle-jerks could not be obtained. Both plantar reflexes gave a flexor response and the abdominal reflexes were readily elicited, and equal on the two sides. Kernig's sign was still present in both legs. On the left side the fundal veins were distended, the arteries were small and difficult to see, and the whole fundus appeared hazy; the edges of the disc were blurred and œdematous; the swelling measured more than 1 and less than 2 D.; no hæmorrhages were seen. On the right the veins were full, the arteries normal, and except for some slight blurring of the inferior and temporal quadrant the edges of the disc clear. All movements of the right eye were normal. The left third cranial nerve was completely paralysed. The left pupil was widely dilated and did not react to stimulation with intense light, the right pupil was small or of moderate size in dull light, and reacted sluggishly to intense light and doubtfully also on accommodation. The lower half of the right side of the face was paretic. The other cranial nerves were unaffected. The blood-pressure by Riva Rocci apparatus in the right brachial artery was 110 mm., in the right leg 114 mm., and in the left brachial artery 109 mm.

On June 21 the patient had had a slight convulsive seizure lasting about twenty seconds, during which time urine was voided. About 12.50 p.m., on June 24, the patient was observed to have developed stertor: no movements occurred. Five minutes later on examination he showed ptosis of both eyes of about equal severity and a bilateral divergent strabismus. The left pupil was dilated and fixed, the right of pin-point size and also fixed. Both arms and both legs were flaccid. The left knee-jerk was readily elicited, whilst the right could not be obtained, and both plantar reflexes gave a flexor response.

Three minutes later (12.58 p.m.), both arms were stiff, the right more so than the left. A knee-clonus was readily elicited on both sides, and both plantar reflexes gave an extensor response. By this time the breathing was

regular and deep. At 1.1 p.m. stertor again developed and was accompanied by flexor-extensor movements of the right half of the body and of the right arm and right leg. The drooping of the upper eyelids previously noticed disappeared and the right eyeball now looked directly forwards, whilst the left still deviated outwards. Lumbar puncture was now performed (1.5 p.m.) and two test tubes full of deeply blood-stained fluid obtained. For a time this procedure seemed to give relief, but at 1.30 p.m. stertor again set in and the patient became cyanosed. For the first time since admission the palpebral fissures were now equal, but the visual axes of both eyeballs deviated widely temporalwards. The pulse was small and thready. Gradually respiration failed, the respiratory movements becoming more and more feeble and shallow in spite of the administration of oxygen. The heart was heard beating ten minutes after all apparent respiratory movements had ceased. Artificial respiration was begun, but death occurred at 2 p.m.

At the *post-mortem* examination, which took place on June 25 (P.M. 583 1911), an aneurysm measuring 1·5 cm. by 1 cm. was found at the junction of the left internal carotid and left posterior communicating arteries. This aneurysm had ruptured and a mass of blood-clot measuring 3 cm. in diameter was found over the anterior and internal subjacent portion of the left temporo-sphenoidal lobe. The meninges around the circle of Willis, more especially in the posterior fossa, were densely matted. The left third cranial nerve was stretched over and was adherent to the posterior extremity of the aneurysm. The lepto-meninges everywhere were stained brown. The lateral ventricle on both sides contained recent red clot and the surfaces of the spinal cord, more especially the posterior, were covered with dark red blood-clot. The brain weighed 1,403 grm.

The cerebral arteries appeared healthy. The heart weighed 269 grm. The valves appeared healthy; on the pericardium a few milk-spots were seen. A few fatty streaks were seen in the lower thoracic and in the abdominal aorta and at the bifurcation of the common carotid artery. The crypts of the tonsils contained muco-pus and each tonsil measured 2·3 cm. in diameter. The liver, lungs, and kidneys were congested. In the left kidney a small congenital cyst was found, and on the surface of the spleen was a small patch of perisplenitis. A small calcareo-caseous nodule was present in one of the cæcal glands.

Thus, apart from the cerebral aneurysm which had ruptured and caused death, all the organs appeared healthy. There were no signs of any cardiovascular hypertrophy, of old or recent endocarditis, and nothing to suggest any syphilitic infection.

A case of death from cerebral hæmorrhage which showed at autopsy a ruptured aneurysm at the junction of the left anterior cerebral and anterior communicating arteries and an unruptured aneurysm at the junction of the right carotid and posterior communicating arteries, with great cardiovascular hypertrophy, general arterial degeneration and atheroma of the cerebral

arteries, in which manifestations of intracranial disorder first occurred at the age of 47. Three apoplectiform attacks preceded the fatal issue. Headache, noises in the head, double optic neuritis, paralysis of the left external rectus, ptosis left. Death from respiratory failure fifty days after the onset of acute symptoms.

Case 4.—43036/07. Matilda H., married, aged 49, was admitted to the London Hospital under the care of Dr. Francis Warner on December 14, 1907, and died on December 30.

Until 1905 her health had been good, but about two years before her death "family matters went wrong," she began to worry, became subject to attacks of severe frontal headache and suffered greatly from noises in the head and sleeplessness. In the months preceding her admission the headaches had been more frequent, more persistent and more severe. On the afternoon of November 10, 1907, fifty days before death, whilst sitting at home in her kitchen she had a "seizure"; the attack came on suddenly: "a feeling of fulness came before my eyes and was then followed by a splitting headache and severe giddiness: my sight became dim and misty, and everything looked indistinct and far away." She did not completely lose consciousness nor did she vomit. During the attack she was able to move her limbs naturally and at will, and could understand what the people around her were saying. The "seizure" lasted about five minutes; it was followed by a feeling of lassitude and inability to stir, which lasted until bed-time but did not prevent her carrying out her household duties. On the morning of the 11th she got up at the usual time and prepared breakfast, but complained of severe vertical headache; this headache persisted for three days. On November 13 she began to see double, and it was noticed that her left upper eyelid drooped; but at this time her limbs were not affected. On the 14th, four days after the seizure, she did the weekly washing for the household.

Two weeks later, on November 28, whilst at work in the washhouse, "a feeling of giddiness seized me: I sat down on a chair, then I completely lost myself and fell on to the floor: when I came to myself I was in bed; then I vomited incessantly for several days: the vomiting bore no relation to the intake of food and did not relieve the headache." She stayed in bed for twelve days. When she got up her memory for recent events, for instance as to where she had put her purse or placed things about the house, had become extremely bad. From that time until admission she complained of a constant severe headache located at the back of the eyes and over the vertex, of diplopia for distant objects, and of a dislike for bright lights. The headache was worse when the patient lay recumbent; it did not prevent sleep and was always worse shortly after waking. The ptosis of the left eye persisted unchanged.

The patient was a married woman of 49; her periods had been irregular for the six months preceding admission. Her husband, aged 51, was alive and, except that he was subject to attacks of acute gout, healthy. Her father, aged 85, was living and healthy; her mother died at the age of 65 from "asthma and bronchitis." She was the fourth of six children, her three brothers and two

sisters were alive and healthy. She married at the age of 18 and had had fourteen children and two miscarriages: (i) male, living and healthy, aged 30; (ii) miscarriage at five months; (iii) female, living and healthy, aged 28; (iv) male, living and healthy, aged 26; (v) full-time child, born dead; (vi) male, living and healthy, aged 24; (vii) male, living and healthy, aged 23; (viii) male, lived three days; (ix) male, living and healthy, aged 21; (x) female, died, aged 2; (xi) female, living and healthy, aged 15; (xii) male, died, aged $1\frac{3}{4}$; (xiii) female, living and healthy, aged 12; (xiv) miscarriage of twins at $3\frac{1}{2}$ months; (xv) male, living and healthy, aged 10; (xvi) female, died, aged 8 months, of "wasting." There was no history suggestive of venereal infection.

The patient was a well-covered woman of healthy appearance. Mentally she was dull and apathetic. Speech was unaffected. Memory for past events was clear and she gave her history in clear, lucid terms; she volunteered the information, however, that her memory for recent events was poor. She suffered neither from hallucinations nor delusions. She complained of a constant severe headache at the back of the eyes and over the vertex, which was most severe on waking and in the early morning. The headache did not extend into the neck or temporal regions and was not accompanied by pressure or percussion tenderness of the skull. She complained of a constant feeling of sickness which occasionally led to actual vomiting; the vomiting did not relieve the headache or the nausea, and bore no relation to the intake of food.

There was no paralysis of any limb, and to tests no alteration in sensibility could be discovered.

The knee-jerks were readily elicited and equal; ankle-clonus was not obtained. The deep reflexes of the upper extremities were normal. On each side stimulation of the sole of the foot gave rise to a brisk response; the first response was usually extensor, but then the toes would flex.

The left palpebral fissure was narrower than the right, and the left upper eyelid could not be raised voluntarily. For objects at a greater distance than 18 in. from the eyes, double vision occurred, for nearer objects single vision. The right external rectus was paralysed; all the other muscles moving the eyeballs acted normally and nystagmus was not present. The pupils were equal and reacted well both to light and to accommodation. There was some evidence of left facial paresis: the left nasolabial fold was less definite than the right, and the whole of this half of the face appeared flat; when the patient was requested to show her teeth, however, the lips moved equally on the two sides. The tongue was protruded straight and showed no tremor. The movements of the palate and larynx were normal.

The upper edge of the right optic disc was blurred and swollen: the swelling extended over the fundus on the inner side: the veins appeared normal. On the left side there was some œdema around the disc and indefinite patches of white exudate were seen on the inferior aspect of the fundus; the line of the inferior temporal artery around the disc on this side could not be traced: the other arteries were small, but on this side the veins were all slightly enlarged. The intra-ocular tension of both eyes was high. The patient

complained of severe pain in the back of the eye, made worse by light. Vision with each eye was fair and the visual fields to coarse testing not grossly impaired.

Hearing was normal, but the patient complained of a noise as of "water rushing through my head." The drums appeared healthy.

The lymphatic glands were not enlarged. A heaving impulse was felt in the fifth space in the mid-clavicular line. The second cardiac sound at the aortic area was accentuated, the first sound at all areas normal and there were no bruits. The lungs were unaffected and the chest moved normally. The wall of the radial artery could be felt, but was not abnormally thick. The blood-pressure was not greatly raised. The liver and spleen could not be felt and to percussion were not enlarged. The patient was constipated. The quantity of urine passed in twenty hours varied from 12 to 44 oz. The urine contained neither albumen nor sugar and casts were not present.

The rate of the pulse varied from 68 to 76 per minute, the rate of respiration from 20 to 24 per minute. The temperature throughout was raised; on admission it was 99·8° F., before death it attained the height of 103° F.

For eight days after admission the patient appeared to improve, her headache became less severe and her mental condition brighter. On December 22, a relapse set in: she became comatose, and passed urine and fæces incontinently into the bed, whilst the rate both of the pulse and of respiration rose. Venesection on the 24th led to no improvement in symptoms or general condition. Death occurred from respiratory failure on the morning of the 30th, seventeen days after admission and seven weeks after her first seizure.

An autopsy was performed on December 31 (P.M. 1181 1907). A ruptured aneurysm, of the size of a pea, was found at the junction of the left anterior cerebral and anterior communicating arteries. Around this aneurysm old and recent haemorrhage into the meninges had occurred. The recent haemorrhage had caused laceration of the brain substance and caused a rupture into the right lateral ventricle. A second, unruptured aneurysm of about the same size was found at the junction of the right carotid and right posterior communicating arteries. The brain weighed 1,474 grm. The left carotid artery at its point of bifurcation was slightly dilated. The heart generally, and more especially the left ventricle, was hypertrophied. Weight of heart 453 grm. As evidence of atheromatous changes a few minute fatty plaques were observed in the intima of the basilar and right posterior cerebral arteries; a few fatty plaques in the wide coronary arteries; fatty flecks and streaks in the carotids; three large fatty, slightly calcareous buttons and numerous fatty, slightly calcareous plateaux and flecks in the aorta and a fatty patch in the aortic cup of the mitral valve. The intima of the femoral arteries was hypertrophied and the media of these vessels showed evidence of slight degeneration. The kidneys were atrophic and together weighed 270 grs. The lungs showed atrophic emphysema with hypostatic congestion.

Under the microscope the wall of the aneurysm on the anterior cerebral artery was seen to consist of fibrous and a few small shreds of elastic tissue:

no muscle was seen. The anterior cerebral artery nearer its origin showed small thickenings of the intima composed of fibrous tissue and elastic fibres; the elastic fibres appeared to have been developed from the elastic stripe. In the site of the swelling at the bifurcation of the left carotid artery the media contained no elastic fibres; the swelling itself was formed partly of muscularis and partly of fibrous tissue.

There was a general hypertrophy of the intima of the aorta; this hypertrophy was found chiefly internal to the stripe and consisted of an inner dense fibrous and an outer fibro-elastic layer. At the site of the flecks the intima was further thickened and here formed nodules. The nodular thickening occurred internal to the elastic stripe. The external portion of this thickening was occupied by atheromatous débris, which was in part calcified. The layer of longitudinal muscles and elastic was not involved in the degeneration. The structure of the buttons was essentially similar. The media everywhere showed an excess of fibrous stroma. The intima of the femoral arteries was hypertrophied. A longitudinal layer of muscles was recognized between the elastic lamina and the stripe. Internal to the elastic stripe was a zone of concentric elastic fibres. The media contained many small spindle-form areas of degeneration.

A case of aneurysm of the right intracranial carotid artery close to the junction with the right middle cerebral, which led to death from cerebral hæmorrhage after giving rise for thirteen months to chronic headache. The first leakage apparently occurred whilst the patient was at stool and was accompanied by acute headache, vomiting, pains in the abdomen and legs. Ptosis of the right upper eyelid appeared fourteen days subsequently and on the sixteenth day a second leakage was ushered in by a convulsion and was followed by acute delirium. He was admitted to hospital on the twenty-first day and at that time showed complete paralysis of the right third cranial nerve with ptosis, optic neuritis, Babinski's sign on the left side, mental disorientation, free blood in the cerebrospinal fluid and albuminuria; under observation the reflexo-motor signs varied. Death occurred from respiratory failure on the thirty-sixth day after the onset of the acute manifestations.

Case 31.—30115 08. Charles E., aged 40, grocer, single, was admitted to the London Hospital under the care of Dr. W. J. Hadley on January 16, and died on February 1, 1908.

The patient was a slight, well-covered man of 40, with dark hair and grey beard, who looked somewhat older than his years. The history of his previous health is somewhat uncertain, as at the time of admission his mental condition was curious and he had no idea of either time or space.

He stated that his health had been uniformly good until the age of 38, two years before admission; he then suffered from an attack of "appendicitis," for which he was operated upon at St. Bartholomew's Hospital, E.C. After

this operation for eight months his health was fair. He then became subject to recurring attacks of occipital headache; the attacks were not severe enough to keep him from work. He denied all venereal infection and had not been addicted to alcoholic excess. For many years he had been deaf in his right ear in consequence of an attack of otitis media in childhood. He was an only child. There was no family history of vascular or nervous disease.

At 11.30 p.m. on December 27, 1907, whilst at stool, he was seized with severe abdominal pain, "shooting down my back and into my legs"; he passed a large stool and then "an electric shock seemed to run up my back into the top of my head." He managed to walk indoors but seemed "dizzy and queer." For half an hour he suffered from an intense headache and then vomited a large black vomit: the vomiting eased the pains in his head. He went to bed and immediately fell asleep. On the morning of the 28th he felt ill: a doctor was called in, who found some paresis of the patient's left leg. Between December 28 and January 16, when he was admitted, he complained of constant, severe pain in the lower part of his back, and of weakness of the legs. On January 10, ptosis of the right eye was noticed. On January 11, he suffered from a seizure which lasted for about fifty-five minutes: it came on suddenly, and in it he was "stertorous, cried out, and became stiff all over." The seizure itself was followed by shrieking and violent delirium, for which he was injected hypodermically with morphia. On the four succeeding nights he was delirious, and on the night of the 15th to 16th he had "five attacks with delirium and excitement."

On *admission*, twenty days after the onset of the illness, he made few complaints: he lay comfortably in bed and talked freely. He answered questions readily and volunteered sensible information. He had little idea of time or space; four hours after admission he stated that he had been in hospital three days: attempts to rectify this impression received the reply, "Well, I've been here three days, I know."

Speech was unaffected, and throughout his stay in hospital he did not vomit.

On the left forearm, just above the head of the radius, was a healing abrasion, brought about in his violent struggles when delirious, and over the sacrum were patches of reddened and roughened skin. The left knee was swollen, tender and slightly reddened, and the joint contained excess of fluid. The skin over the left heel was abraded. There was no anæmia and the lymphatic glands were not enlarged. On admission the temperature was 97° F., the rate of pulse 64 per minute, and the rate of respirations 30 per minute.

There was no absolute paralysis of any muscle, yet the patient was unable to turn himself in bed. All voluntary movements were carried out without precision in a clumsy, jerky fashion. The tone of the muscles of the extremities was normal and no abnormal muscular twitchings were observed. Spontaneous movements of any sort rarely occurred and under observation the left leg was never moved. He complained of no spontaneous pains and

to testing with cotton wool, the prick of a pin and for the recognition of passive movement no sensory loss was discovered.

Neither knee-jerks nor ankle-jerks could be elicited. On the right side the plantar response was flexor, on the left an extensor response was obtained. Kernig's sign was not present on the left, on the right it was doubtfully obtained.

The vessels of the optic fundi were full and some œdema was seen along their course; the edges of the discs were sharp and no hæmorrhages were observed. On the right side he could neither appreciate the ticking of a watch nor the sound of a tuning-fork applied to the mastoid; both drums appeared normal. Smell and taste were unaffected.

The right upper eyelid drooped and could not be raised voluntarily, the left lids moved normally. The right external rectus muscle acted well, but the remaining muscles moving the right eyeball were completely paralysed; on the left side all ocular movements were normal. Nystagmus was not present. The right pupil was widely dilated and did not react to light or on attempted movements of this eye; the left pupil was moderate in size and reacted normally to light and on accommodation. The facial movements were normal. The tongue was protruded straight and held steadily. The movements of the palate and larynx were unaffected.

On admission the actions of the bladder and rectum were controlled and the movements of the spine were unaffected.

The apex-beat of the heart was felt in the fifth space just internal to the mid-clavicular line; the cardiac sounds were clear. There was no abnormal dulness over the lungs, and the breath sounds were normal.

A well-healed scar, 4 in. long, was seen over the right iliac fossa. The liver and spleen could not be felt, and to percussion there was no evidence of any enlargement of these organs.

The quantity of urine passed in twenty-four hours varied from 24 to 64 oz., and the specific gravity from 1021 to 1028. The urine was acid, and, on boiling, yielded a thick cloud of albumin; blood and sugar were never found. On standing, a quantity of floating mucus was deposited; this deposit consisted in the main of broken-down pus cells and squamous epithelial cells, but in it occasional hyaline casts were seen.

On January 18 lumbar puncture was performed. Cerebrospinal fluid escaped under considerable pressure. This fluid was bright red in colour and uniformly stained with blood. On microscopic examination many red cells and a few polymorphonuclear leucocytes only were found; bacteriologically the fluid was sterile.

For a few days after admission the patient's general condition improved. The headache became less severe, and his mental condition clearer; he now complained of a sensation "as if I were falling backwards." On the nights of the 17th, 18th, and 19th he was delirious; he called to imaginary persons, and received imaginary answers, but in the daytime he was more or less rational in his behaviour, though in general he lay with his eyes closed, complaining

of little and speaking only when addressed. He was now able to carry out certain voluntary movements, but when left to himself performed few.

The physical signs of disease varied from day to day and from hour to hour. Sometimes on the right side a knee-jerk could be obtained, at others it was absent; at still others it was grossly exaggerated; the left knee-jerk also varied, and occasionally was absent. The plantar responses also showed variations; sometimes the right would be extensor, at others flexor; the response on the left side varied similarly, but not synchronously. The mental state gradually improved between January 19 and January 30; spontaneity returned, headache was no longer complained of, the sensation of falling backwards disappeared, and the nocturnal delirium ceased.

In the early hours of January 30 the patient was seized with severe headache; the headache was at first chiefly frontal, but soon spread backwards over the occiput and into the neck. It was accompanied by pressure tenderness of the scalp. There was, however, no rigidity of the muscles of the neck or of the back. The onset of the headache was not accompanied by any corresponding alteration in the rate of the pulse or of respiration.

At midday, on the 30th, he suddenly became worse and lost consciousness; breathing became shallow, and occasionally stertorous. The right pupil remained dilated, but the left contracted to the size of a pinhead. The rate of the pulse remained about 100 per minute.

Lumbar puncture was now performed, and cerebrospinal fluid under considerable pressure escaped in a steady stream. The fluid was fairly clear, and intimately mixed with bright red blood; two ounces were collected. The breathing became deeper and more regular, and a movement of the right hand to the head, apparently of a voluntary nature, was observed. Consciousness of his surroundings, however, did not return. Frequently repeated flexion-extension movements of the right thigh were now noted.

At 9.30 p.m. on this day he lay flaccid and unconscious. The left pupil was small, the right dilated, but not to such a great extent as heretofore. There was definite unsteadiness of the eyes on looking outwards of a nystagmoid nature. The right eye showed much greater freedom of movement than had been previously observed. Exposure of the eyes to light irritated the patient, and was followed by movements of the head and general restlessness of the body. At this time breathing was regular, and expiration was accompanied by a flapping of the cheeks and a short groan. Flexion of the right thigh was frequently seen: all the other extremities lay flaccid by the trunk. The knee-jerks could not be elicited. On the right side there was a ready extensor plantar response; on the left no plantar response could be obtained. The wrist- and elbow-jerks were absent. On the right side Kernig's sign was positive, on the left negative. No reaction to painful pressure or to the prick of a pin could be obtained from any portion of the head, trunk, or extremities. The right half of the face in both its upper and lower portions was completely paralysed, and the ptosis of the right upper lid persisted.

Eight hours later the patient's condition again became worse. Lumbar puncture was again performed, and eleven drachms of bright red fluid were obtained. After this he became steadily weaker, the breathing took on the Cheyne-Stokes' characters, and the respiratory passages filled with mucus.

Death from respiratory failure occurred twenty-four hours later at 8.30 a.m. on February 1, 1908, thirty-six days after the initial seizure.

At the autopsy performed on the afternoon of the day of death only an examination of the head was permitted. A small ruptured aneurysm, of the size of a haricot bean, was found on the right intracranial carotid artery close to the origin of the middle cerebral artery. The right temporal convolution as far as the second temporal sulcus was almost completely destroyed. Over the base of the brain and over the vertex and the region of the Sylvian fissure on the right side there was considerable subarachnoid and subpial hæmorrhage, and around the aneurysm there was a considerable quantity of older blood-clot. The lateral and fourth ventricles were distended with blood-stained fluid. Otherwise the cerebral arteries appeared quite healthy, and showed no sign of inflammation or atheromatous change.

A case in which the autopsy revealed a ruptured and partially thrombosed aneurysm of the left middle cerebral artery near the base of the brain, considerable atheroma of the cerebral arteries, severe general arterial degeneration, and slight cardiovascular hypertrophy in a woman aged 67. Eighteen years before death this patient, after complaining of left-sided local headache, had suffered a right hemiplegia with motor aphasia from which she almost entirely recovered. Seven months before death she developed ptosis and supraorbital neuralgia on the left side, the pains of which for five months were relieved by an injection of alcohol. Eighteen days before death a sudden apoplectiform seizure occurred with development of motor aphasia, œdema of the soft tissues around the left eye, complete paralysis of the left third cranial nerve and ptosis and noises in the ears; partial recovery. Seven days before death, after a seizure, paralysis of the right face, tongue, and arm, and severe lethetude appeared. Final seizure and respiratory failure on day of death.

Case 14.—40432/1913. Jane P., aged 67, married, was admitted to the London Hospital under the care of Dr. W. J. Hadley on February 11, and died on February 23, 1913. In 1891, at the age of 45, after suffering for several years with chronic left-sided headache, she had a seizure which was followed by a right-sided "hemiplegia and motor aphasia"; at this time she was "unable to talk" for fourteen days, and in bed for six weeks. The right hemiparesis completely disappeared, and except that she was still subject to recurrent episodical attacks of severe pains in the head, for the next twenty years her health was good. In February, 1912, she suffered an attack of erysipelas. In June, 1912, she was in-patient at the Croydon Hospital, complaining of trouble in opening her left eye and intense supraorbital

neuralgia. For the neuralgia, alcohol was injected into the left supraorbital nerve. After the injection she was never able to raise her left upper eyelid, but the pain was relieved. About Christmas, 1912, the left supraorbital pains returned, and on December 31, 1912, the supraorbital nerve was again injected with alcohol; this injection, however, yielded no relief. On February 3, 1913, she suffered from a seizure in which consciousness was lost; on the return of consciousness she was unable to talk; she knew what she wanted to say, could write and draw, but could not utter the words. On February 4, 1913, speech returned. She then became subject to nocturnal delirium, and her ideation became inconsequent. After the seizure until admission she was kept in bed.

On admission: The tissues around the left orbit and in the left temporal region were œdematous and pitted slightly on pressure. Cerebration was slow and ideation defective. In general she lay in bed in a dazed and drowsy condition taking little interest in her surroundings. At night she was restless, noisy, and made attempts to get out of bed. To questioning she answered rationally and quickly. Speech was not affected. She was well orientated and delusions were not present. Memory was defective.

She was completely unable to open the left eye and the left upper lid drooped. The external rectus and superior oblique muscles acted normally, but the power of movement by all the muscles supplied by the left third cranial nerve was extremely feeble. The left pupil was dilated and did not react either to light or attempted convergence; the right pupil was of moderate size and reacted normally. The movements of the jaws, face, palate, tongue, and larynx were unaffected. There was considerable sensory loss over the area of skin supplied by the left supraorbital nerve, probably consequent on the injection given on December 31, 1912: hyperalgesia and loss of sensation were not discovered elsewhere in the skin supplied by the fifth cranial nerve. Hearing was unaffected, but the patient complained of a "roaring noise in both ears": the drums appeared healthy. The optic discs and ocular fundi on both sides appeared healthy. Smell and taste were unaffected.

There was no motor paresis of the limbs or trunk, and the tone of the muscles of the limbs was normal. No sensory disturbance over the body or extremities was discovered.

The knee-jerks and ankle-jerks were not exaggerated, and both plantar reflexes gave a flexor response. Neither upper nor lower abdominal reflexes could be obtained. The wrist- and elbow-jerks were normal and a jaw-jerk could be obtained.

The action of the sphincters was controlled.

The chest was emphysematous and the area of cardiac dulness small. A feeble cardiac impulse could be felt in the fifth space just inside the mid-clavicular line. The cardiac sounds were normal. The wall of the radial pulse could just be felt but the blood-pressure was not unduly high. No abnormal signs were discovered in the abdomen. The urine contained a cloud of albumin, but no sugar; its specific gravity varied from 1017 to 1025. The patient had suffered from recurrent attacks of epistaxis, but no abnormal

appearances were detected in the nose, and all the sinuses on illumination appeared normal.

On the second evening after admission she was noisy, made irrelevant remarks and suffered from hallucinations and illusions. About 10.30 p.m. on this day she suddenly fell back in bed; her breathing became stertorous and dyspnœic; she lost consciousness, and for two or three minutes moved her limbs violently. After the seizure she was very irritable and restless, and yet could not move her limbs voluntarily. The puffiness around the left orbit increased. On examination complete paralysis of the left third cranial nerve, weakness of the lower half of the right face and of the right half of the tongue, and a monoplegia of the right upper extremity with increased reflexes were discovered. The right knee-jerk was more brisk than the left, but ankle-clonus was not present and both plantar reflexes gave a flexor response. On the morning of the 13th speech was impaired, but the lower extremities and left arm were under voluntary control; and the patient suffered from retention of urine with overflow. She could understand written and printed words, and attempted to answer the questions. During the whole of the 14th, respirations were of the Cheyne-Stokes type. On the 15th she talked freely but her remarks were inconsequent and nonsensical. From this time until the 21st her condition altered little: on this day she again suddenly lost consciousness and died from respiratory failure.

At the autopsy, P.M. 140/1913, performed on February 24, a ruptured and partially thrombosed aneurysm, 3 cm. in diameter, was found on the left middle cerebral artery near the base of the brain. No atheromatous changes were seen in the aneurysm itself, but the cerebral arteries generally were wide and showed severe atheromatous changes. The left third cranial nerve was small and grey in colour. The meninges in the neighbourhood of the aneurysm and in the right Sylvian fissure were deeply stained with altered blood-pigment and much matted. The myocardium everywhere was fatty. The wall of the left ventricle was 2 cm. in thickness. The base of the mitral and aortic cusps was slightly thickened. Fatty plaques and flecks were observed throughout the thick-walled aorta and calcareous plaques were seen in the aortic arch and abdominal aorta. The coronaries were wide and showed many thick-set fatty streaks and plaques, whilst the carotids were studded with fatty streaks. In the posterior parts of both lungs evidence of purulent bronchitis and hypostatic broncho-pneumonia was discovered. The kidneys were "granular:" in colour they were red, and their surfaces were irregular and showed subcapsular nodules. The cortex was narrowed, and owing to the presence of large arcuate arteries showed a tortuous pattern.

In *nine* other cases multiple seizures from partial ruptures of a cerebral aneurysm occurred.

Case 23 (p. 284) was that of a grocer, aged 41, who had been under more or less continuous observation at hospital over a period of sixteen years, and had complained of recurring attacks of headache, and been

blind of an anæsthetic left eyeball for three years. The *post-mortem* examination showed that he died from the rupture of an aneurysm of the size of a currant-grape at the junction of the right anterior cerebral and anterior communicating arteries. In this patient the first bleeding occurred forty-three days, a second twenty-five days, a third seven days, and a fourth twenty-four hours before death in coma.

Case 22 (p. 287) was an example of multiple leakages and seizures due to the rupture of a small aneurysm of the left posterior cerebellar branch of the basilar artery. At the autopsy a mass of blood-clot which had pressed upon the pons, cerebellum, medulla, and the occipital lobe of the left cerebral hemisphere was discovered. The patient was a married woman, aged 42, who, when aged 25, and 8 months pregnant of her second child, had suffered a transient hemiplegia affecting the right face, tongue, arm, and leg, and who for the five months preceding death had complained of slight epileptiform (*petit mal*) attacks with visual auræ. The first evidence of definite leakage was a seizure nineteen days before death, followed by a second twelve days and a third ten days before the fatal issue. On admission to hospital the patient was delirious, and showed paresis of the left half of the face, right hemiplegia, affecting the arm and leg, and complained of tenderness in the left occipital region and stiffness of the neck; the head was held in the left cerebellar attitude, but no nystagmus was observed; in the right eye early optic neuritic changes were present.

Case 16 (p. 289) at autopsy showed a ruptured aneurysm, of the size of a pea, at the junction of the left anterior cerebral and anterior communicating arteries. The patient, a carpenter, aged 53, sustained a first seizure whilst watching a football match twenty-seven days before death. This seizure was followed by a state of delirium and a complaint of headache and cerebral vomiting; later he began to suffer from epileptiform attacks, and became clumsy in all his movements. Five days and three days respectively before death he sustained a second and a third seizure; after the third seizure he was admitted to hospital, where he died.

In Case 29 (p. 292) the rupture of an aneurysm, of the size of a pea, on the left anterior cerebellar artery caused the death of a young man, aged 22. Six days before death he sustained his first seizure, followed by a state of mental instability. Three days later a second seizure led to drowsiness, left facial paralysis, bilateral corticospinal interference, left cerebellar posture of the head, stiffness of the neck, and the appearance of free blood in the cerebrospinal fluid, and to death without any recovery of consciousness.

Case 3 was that of an upholsterer, aged 60, who died from the rupture of an aneurysm of the size of a pea on a central branch of the right middle cerebral artery. At the autopsy this patient also showed a smaller, unruptured aneurysm on a second branch of the same vessel, severe atheroma of all the cerebral arteries, severe general arterial degeneration, and great cardiovascular hypertrophy. Two years before death this patient had suffered a monoplegia of the left lower extremity, from which he soon entirely recovered; fifty hours before death a second seizure led to paresis of the left arm, abolition of the right corneal reflex, and a state of deep coma, from which he never recovered. After admission to hospital he suddenly became convulsed; after the convulsion the left-sided paresis had increased, and signs of left cortico-spinal interference appeared.

Case 12 was that of a woman, aged 46, who was admitted to hospital in an irritable condition after a seizure, which had come whilst she was minding her fruit stall in the street, complaining of some subjective weakness of the left upper extremity, right-sided headache, leading to nausea and vomiting, and giddiness, but showing no optic neuritis or other sign of intracranial affection. Three days after admission all her symptoms had cleared, and on the fourth day she discharged herself, to be readmitted on the fifteenth day after the initial seizure in a deeply unconscious state, which was again said to have come on suddenly, and from which she never recovered.

At the autopsy a ruptured aneurysm was found on the rightmost of three anterior cerebral arteries just beyond the origin of the anterior communicating, of the size of a pea. The convexity of the right cerebral hemisphere was covered with old hæmorrhagic clot, and in the arteries generally there was a moderate grade of degeneration and a moderate degree of cardiovascular hypertrophy.

Case 15 was that of a married woman, aged 60, who six months before death suffered from an attack of "inflammation of the lungs," after which she never recovered her health, but became subject to recurring attacks of severe headache. The initial seizure in this case occurred whilst the patient was in bed on the seventy-fourth night before death; she woke with a sudden severe headache, leading to vomiting. This attack of headache only lasted one day, but twenty-six days later she became "dazed" after a "bad shivering attack," and in this condition was admitted to hospital. This patient showed numerous scars on the shins, apparently due to old varicose ulcers, for her serum reacted negatively to the Wassermann test. The blood-pressure in the

arm measured 188 mm., and the urine contained a trace of albumen. She complained of severe frontal headache, but the fundi appeared healthy. The left face showed a slight upper motor neuronic weakness, otherwise no affection of the cranial nerves was detected. Motion, sensation, the reflexes, and the action of the sphincters were unaffected. Despite the negative Wassermann reaction the condition of this patient was thought to have a syphilitic basis, and she received an injection of 0·6 grm. neosalvarsan. After this injection the headache became more severe and more persistent, and she began to vomit several times daily. Twelve days after the injection insomnia, nocturnal delirium, "mental" incontinence of urine and fæces, and delusions, made their appearance, and the paresis of the left face became more noteworthy, whilst the quantity of albumin in the urine increased. These manifestations altered little until the day of death. On that day, whilst sitting in a chair in the ward, she suddenly fell backwards, and a few minutes later died from respiratory failure. At the autopsy all the large basal cerebral vessels were dilated, and on the right middle cerebral artery was an aneurysm of the size of a cherry-stone, which had ruptured. In the great vessels was evidence of severe arterial degeneration, and in the cardiovascular system generally some slight hypertrophy.

Two years before death a packing-case maker, aged 36 (Case 25), who for many years had been addicted to alcoholic excesses, suffered a "fainting attack, after which he was never the same man." At the time of the attack, and for three weeks afterwards, he complained of an unbearable pain in the head, which gradually became less severe, but never entirely ceased. Twelve hours and a half before death this patient was at work, when he suddenly shouted that the headache had returned, and that he felt as if the top of his head would burst; then he became giddy, seemed to lose himself, and for the next hour and a a half struggled and threw himself about. On admission he was deeply comatose and stertorous. At the autopsy an aneurysm of the size of a cherry-stone, which had ruptured, was found on the left intracranial carotid artery. In the heart and vessels few signs of disease were present, but the wall of the aneurysm itself showed atheromatous changes.

The last of this class is Case 26, that of a married woman, aged 36, who during the two preceding winters had suffered from chronic bronchitis. Three months before death she began to complain of episodical attacks of severe left frontal headache. Three days before death she was out shopping when she suddenly "came over giddy,"

complained of severe left temporal headache, fell in the street and was taken home and put to bed. Throughout the next three days the headache persisted, and she remained in bed at home. On the night of her death she was found deeply unconscious, having vomited, showing proptosis of the left eye and widely dilated fixed pupils, whilst the knee-jerks were abolished and both plantar responses were extensor. At the autopsy three aneurysms were found on the cerebral vessels, a ruptured aneurysm of the size of a grape-stone at the junction of the left intracranial carotid and posterior communicating arteries and two unruptured aneurysms on the right middle cerebral artery. The more proximal was the smaller and lay in the fork of a primary division of the middle cerebral, whilst the more distal, a little larger than a grape-stone, lay slightly nearer the base of the brain. Atheromatous changes were present in the peripheral portions of the unruptured aneurysms, but otherwise the cerebral arteries appeared normal, and there was only slight general arterial degeneration and no cardio-vascular hypertrophy.

In Thirteen Cases Multiple Seizures occurred at varying Intervals before Death.

Case	Sex	Age	First seizure	Second seizure	Third seizure	Fourth seizure	Fifth
26	♀	36	3 days	8 hours	—	—	—
29	♂	22	6 ,,	2 days	—	—	—
12	♀	46	19 ,,	16 hours	—	—	—
30	♂	19	24 ,,	3 days	2 days	—	—
23	♂	41	25 ,,	7 ,,	Few hours	—	—
16	♂	53	27 ,,	5 ,,	3 days	2 days	Few minutes.
31	♂	40	36 ,,	21 ,,	8 hours	—	—
4	♀	49	50 ,,	33 ,,	8 days	—	—
15	♀	60	74 ,,	48 ,,	Few minutes	—	—
25	♂	36	2 years	12 hours	—	—	—
3	♀	60	2 ,,	50 ,,	—	—	—
22	♀	42	17 ,,	6 months	5 days	—	—
14	♀	67	22 ,,	8 ,,	18 ,,	1 hour	—

Patients in whom abnormal conditions of the vessels are suspected are always warned against the effects of a sudden rise in blood-pressure associated with violent voluntary efforts and acute emotional experience. In this series of cases the importance of this warning is well illustrated. The manifestations of the initial rupture in Case 28, that of a young man aged 22, occurred whilst the patient was competing in a public cycle race, and in Case 16, that of a carpenter aged 53, while the patient was watching a local football match and overflowing with excitement. Case 31 sustained his first seizure whilst straining at stool; Case 5 whilst at work painting; Case 29 when at work in the stable; Case 25 when lifting a heavy packing-case; and Case 30 whilst reaching

upwards to paint the top of a window. Seven examples of rupture whilst in the street occurred: Cases 1, 2, 9, 10, 12 (when minding a street-stall), Cases 24 and 26 (shopping); and in one case (Case 27) the acute manifestations developed whilst the patient was in the bath-room, and in another (Case 17) during a bout of coughing.

Although aneursym is one of the relatively common forms of "intracranial neoplasm," when the tumour is due to changes in the media of the arterial wall it grows slowly and only rarely does it attain any considerable size, hence before rupture signs of increased intracranial pressure—hebetude, headache, vomiting, and optic neuritis—are also rare. In this series of cases *in no single instance* where at autopsy the aneurysm was found to be associated with changes in the media only was there clinical evidence that the aneurysm before rupture had led to the development of *optic neuritis*. In Case 25 the patient long before the final rupture took place was blind of his left eye, but here the whole eyeball, which was anæsthetic, shrivelled. On the other hand, amongst 555 cases collected by Beadles, in 16·4 per cent. signs of "tumour only" were present, and in a further 20·7 per cent. "signs more or less suggestive of tumour" occurred before rupture. Aneurysms of embolic origin without an attendant diffuse septic meningitis have only on extremely rare occasions given rise to signs of "increased intracranial pressure," and in this series optic neuritis in association with embolic aneurysm was not noted, although in Case 9 the left disc appeared slightly swollen and the whole of the fundus on this side had a glassy appearance. Cerebral aneurysms of syphilitic origin are more frequently associated with fundal changes, but in this class of case a reactive meningitis is practically always present, and the evaluation of the relative importance of the meningitis and the aneurysmal formation as the cause of these changes is, and has been, always difficult.

In this series of cases of aneurysms due to changes in the media after rupture had occurred the condition of the optic fundi was noted in 16 cases; in 10 the discs, vessels, and fundi were pronounced healthy, and in 6 abnormalities were described.

In Case 4 (p. 252) the ruptured aneurysm was found at the junction of the left anterior cerebral and anterior communicating arteries. Here the upper edge of the right optic disc was blurred and swollen and this swelling extended over the inner side of the fundus; the arteries and veins appeared normal. On the left side there was some œdema around the disc and indefinite patches of white exudate were seen on the

inferior aspect of the fundus, whilst the line of the inferior temporal artery near the disc could not be traced; the other arteries were small, but on this side the veins were all slightly enlarged.

In Case 16 (p. 289) the aneurysm was found at the junction of the left anterior cerebral and anterior communicating arteries; here the right optic disc and fundus appeared pale and the edges of the disc clearly defined, but whilst the arteries were small the veins were engorged. On the left side the disc was swollen ($1\frac{1}{2}$ D.) and its edges blurred and the veins were more engorged than on the right.

In Case 22 (p. 287) the ruptured aneurysm was found on the left posterior cerebellar branch of the basilar artery, and the patient suffered amongst other complaints from epileptiform attacks with visual aurae. In this case the left fundus was described as of healthy appearance, whilst on the right the veins were engorged, the disc pink in colour, its edges indistinct and blurred, whilst the arteries were unaffected.

In Case 27 an aneurysm on the left intracranial carotid artery, of the size of a pea, had ruptured; just before death the right optic disc was described as healthy but the left was said to be swollen.

In Case 30 (p. 249) an aneurysm measuring 1·5 cm. by 1 cm. was found at the junction of the left internal carotid and posterior communicating arteries. The right optic fundus of this patient sixteen days before death showed engorgement of the veins and some œdema of the lower quadrants, whilst on the left side no changes were detected. On the day of death the veins of the right fundus were full and the edges of the inferior and temporal quadrant of the disc blurred, whilst on the left side the whole fundus appeared hazy and the disc showed definite swelling of about $1\frac{3}{4}$ D.

In Case 31 (p. 256), a ruptured aneurysm of the right intracranial carotid artery close to the origin of the middle cerebral artery of the size of a haricot bean was found; twenty days after the initial seizure the veins of both optic fundi were large, and along their course some œdema was present, but the edges of the disc were unaffected.

Headache.—The headaches associated with the presence and rupture of intracranial aneurysms do not differ from those associated with pathological intracranial conditions owning other causes, and frequently give rise to nausea and vomiting. In general, when a patient complains of localized pain in the head, and a cerebral aneurysm is suspected on other grounds, the aneurysm will be found on the same side of the circle of Willis as the headache (Cases 12, 16, 22, 26, 27 and 30), and if the headache is worse in the frontal or orbital region it is likely that

the affected vessel lies in front of the point of entry of the carotid artery into the cranial cavity (Cases 4, 15, 16, 20, 25 and 26), whilst, on the other hand, the complaint of pain localized to the occipital and upper cervical region renders the affection of a vessel in the posterior cranial fossa probable (Cases 22, 30 and 31).

In six of the 31 cases (19·3 per cent.), episodical attacks of headache were the first clinical manifestations which could be attributed to the development of a cerebral aneurysm; in all these cases at a later date the sac of the aneurysm leaked and before death signs of meningeal hæmorrhage appeared. Case 4, a woman, aged 49, for two years before death was subject to attacks of frontal headache and complained of insomnia and noises in the head. Case 27, a compositor, aged 28, for nineteen months before he came under observation suffered from severe attacks of left-sided headache, and for six months from attacks of giddiness complicating the headache. Case 31, a grocer, aged 40, complained of chronic occipital headache for thirteen months before the final ruptures; whilst Case 15, a married woman, aged 60, during the nine months before death suffered from recurring attacks of headache and during her last ten weeks showed defective cerebration. Case 26, a woman, aged 36, for three months before the final seizure complained of frontal and temporal headache limited to the left half of her head; and finally, Case 18, eight days before admission developed acute headache and nausea; she vomited several times, but not until six days later did any local signs of intracranial interference appear.

The interferences with *general cerebral functions* associated with the presence of an unruptured or of a ruptured cerebral aneurysm are not characteristic. Before rupture the blood-supply of some small portion of either hemisphere may be defective and thus produce symptoms, but this appears to be exceptional. After a partial leakage the affected vessel may become thrombosed and localized cerebral softening may thus be set up, or local destruction and ploughing up of portions of the hemispheres may be caused by the blood which has leaked primarily into the meninges. Sudden hæmorrhages are manifested by apoplectic attacks and the development of a more or less deep state of coma, and if the hæmorrhage be large, death without recovery of consciousness occurs. Smaller leakages give rise to less complete abolition of function, and thus states of hebetude, apathy, drowsiness, inattentiveness, associated with irritability, "strangeness in behaviour," "hysterical attacks," defective ideation, delirium, insomnia, nocturnal psychoses and the like may arise, and, if the surface of the cerebrum

be affected and yet not destroyed, convulsions of all sorts may appear.

Hemiplegia.—The motor affections associated with ruptures of cerebral aneurysms depend upon the site of the lesion affecting the corticospinal connections, and this may be cortical, basal, or pontine. Cortical affections produce more limited manifestations than do affections at other points on the course of the pyramidal tracts. In Cases 3, 7, 15, due to an aneurysm on the middle cerebral artery, an affection of the pyramidal tract in the cortex or subcortex was noted, and in Cases 16 and 22 convulsions of an epileptiform type were associated clinically with the finding of localized damage to the cortex of the cerebral hemispheres. In Cases 3, 14 and 30 the Weber syndrome (paralysis of one third cranial nerve with crossed affection of face and limbs) was present, and in Cases 22 and 29 the pontine type of hemiplegia with facial paralysis and crossed paralysis of the limbs occurred, whilst in Cases 6, 17 and 26 pressure due to the rupture of aneurysm at the base of the brain led to bilateral interferences with the upper motor neuronic connections.

At the base of the brain the cerebral arteries bear close relation to the *cranial nerves.* One would expect, therefore, that when an aneurysm had developed upon one of the basal vessels pressure effects upon one or more cranial nerves would not infrequently occur and might prove of localizing value in diagnosis. Judged by this series of cases, however, cranial nerve affection rarely occurs until the wall of the aneurysm ruptures. In Case 14 (p. 260) alone did both the clinical history and the autopsy reveal changes of old standing in any cranial nerve; in this case the third nerve on the left side was found grey, thin and stretched over the sac of an aneurysm, 3 cm. in diameter, on the left middle cerebral artery: the aneurysm had also pressed upon the left Gasserian ganglion and caused a supra-orbital neuralgia on that side.

In *no case* was any definite evidence, clinical or pathological, of an affection of the olfactory nerves obtained.

In Case 23 (p. 284) in association with an aneurysm at the junction of the right anterior cerebral and anterior communicating arteries was a shrivelled and anæsthetic left eyeball and atrophic, grey, left optic nerve. In no case in this series was there any evidence of hemianopsia or other affection of the optic tracts, but in two cases the one recorded by Byrom Bramwell [7] and the other by Weir Mitchell [26] bitemporal hemianopsia due to the presence of a large saccular aneurysm in the infundibular region occurred.

In cases of cerebral aneurysm after rupture the third pair of cranial nerves are those most commonly involved. Ptosis, an affection of the pupils and even total paralysis of the third cranial nerve on one side, are not infrequent.

On the admission of Case 31 (p. 256) to hospital, the right upper eyelid was dropped and could not be raised, the right pupil was widely dilated and fixed, and all the muscles moving the eyeball supplied by the right third cranial nerve were completely paralysed. At the autopsy a ruptured aneurysm on the right intracranial carotid close to the origin of the right middle cerebral artery was found. Case 30 (p. 249), after a seizure, showed a similarly complete paralysis of all the muscles supplied by the left third cranial nerve; at the *post-mortem* examination this nerve was found to be stretched over and adherent to the posterior extremity of a ruptured aneurysm on the posterior communicating artery close to the bifurcation of the intracranial carotid. The patient in Case 27 was deeply comatose when admitted; in him the right pupil was small, the left widely dilated, and neither pupil reacted to the light. Here the ruptured aneurysm was found on the left intracranial carotid. In Case 26, proptosis of the left eyeball, together with fixity of the pupils, was observed; a mass of blood-clot was found at the autopsy pressing upon the left cavernous sinus; this had escaped from an aneurysm at the point of junction of the left intracranial carotid and posterior communicating arteries. Case 24 was moribund on admission; here the pupils were fixed and dilated and a rupture was found to have occurred from the right posterior communicating artery at its point of junction with the posterior cerebral. Case 23 (p. 284) after the second leakage showed severe ptosis of the right eye in addition to the atrophic left eyeball.

In Case 22 (p. 287), on admission to hospital, the left pupil was larger than the right, but both reacted normally to light; a left facial weakness and right hemiplegia affecting the arm and leg were also present in consequence of the leakage of an aneurysm on the left posterior cerebellar branch of the basilar artery. Cases 10 and 13 were deeply comatose on admission. In Case 13 neither pupil reacted to light and the size of both varied greatly from moment to moment; signs of bilateral corticospinal interference were present and at autopsy a ruptured aneurysm on the left anterior cerebral at its junction with the anterior communicating artery was found; whilst in Case 10 both pupils were immobile, and the right dilated and much larger than the left in a man, aged 55, who was admitted one hour before death; here the

aneurysm which had ruptured was found on the corresponding junctional point of the anterior cerebral and anterior communicating on the right side. Case 4 (p. 252) complained of diplopia, but showed no gross abnormality of the pupillary reactions; here complete ptosis of the left upper eyelid and paralysis of the right external rectus muscles were found consequent on the rupture of an aneurysm at the point of junction of the left anterior cerebral and anterior communicating arteries and the presence of a second unruptured aneurysm at the junction of the right carotid and posterior communicating arteries.

In two cases abolition of the conjunctival and corneal reflexes was of some localizing value. In Case 3 on the right side neither of these reflexes could be elicited, and with this loss was associated a paresis of the left upper extremity owing to the rupture of an aneurysm on the right middle cerebral artery; and in Case 25 the left corneal and conjunctival reflexes were completely abolished and the right considerably diminished in consequence of the rupture of an aneurysm on the left intracranial carotid artery.

Case 14 (p. 260) during the seven months preceding death suffered from a left-sided supra-orbital neuralgia and ptosis; alcoholic injections, which were given into the nerve, relieved the acute pains for a time but the patient soon relapsed. At autopsy a firm mass of blood-clot, which had escaped from the left middle cerebral artery, was found filling the cavernous sinus and pressing upon the Gasserian ganglion. The association of major trigeminal neuralgia with the presence of a cerebral aneurysm cannot be very infrequent, for in two other instances which have come under my notice since this series was closed I have seen autopsies which revealed a similar pathological condition, in one of which before death herpes ophthalmicus developed owing to the involvement of the Gasserian ganglion in the mass of blood-clot.

Facial paralysis, due to an affection of the nerve in its intracranial course, and after its exit from the pons, usually signifies that one of the cerebellar arteries is the source of trouble. In Case 29 (p. 292), at the time of admission a stable-boy, aged 22, showed a complete, peripheral type of left facial paralysis and right hemiplegia; at autopsy a ruptured aneurysm was found on the left anterior inferior cerebellar artery. In Case 22 (p. 287) the lower half of the left face was completely paralysed and the upper half weak, whilst the right arm and leg were spastic and paretic in association with a ruptured aneurysm on the left posterior cerebellar branch of the basilar artery.

Facial paralysis of cerebral origin associated with a more or less

complete hemiplegia may also occur in cases of ruptured cerebral aneurysm. Case 4, on admission, in addition to the paralysis of the left external rectus muscle and ptosis of the right eye, showed slight left facial paresis of cerebral origin; at autopsy two aneurysms were found, a ruptured one at the junction of the left anterior cerebral and anterior communicating arteries and an unruptured one at the junction of the right carotid and posterior communicating arteries. In Case 5 a right hemiplegia of arm and leg and some affection of the left extremities, together with a right facial paresis of upper motor neuron type, appeared in consequence of the rupture of an aneurysm at the point of junction of the left anterior cerebral and anterior communicating arteries.

In Case 7 an old man, aged 86, was admitted to hospital complaining of some difficulty with speech, and showing slight left facial weakness; four weeks later he died, and at the autopsy an unruptured aneurysm on the right middle cerebral artery near the base of the brain was found, and all the cerebral arteries were dilated and atheromatous. Case 15, a woman, aged 60, on admission complained of headache, giddiness, and cerebral vomiting, but the only definite sign of intracranial affection was a slight paresis of the left face, apparently of cerebral origin; at the autopsy, seven weeks later, an aneurysm which had burst was found on the right middle cerebral artery near the base of the brain. Case 30 (p. 249), at autopsy showed an unruptured aneurysm on the left posterior communicating artery; on admission to hospital this patient exhibited a complete paralysis of the left third cranial nerve and grave paresis of the right half of the face.

Deafness, as the consequence of an unruptured cerebral aneurysm, is not common. Case 20, a man aged 47, died in hospital of acute diffuse myelitis. Some months before death this patient suffered an "influenzal attack," which left him subject to severe frontal headache, and deaf of his right ear. At the autopsy an unruptured aneurysm of the size of a pea was found at the point of bifurcation of the right intracranial carotid artery.

Case 31, on admission, in addition to a complete paralysis of the right third cranial nerve, showed an almost total nerve-deafness of the right ear; the aneurysm which had ruptured was found on the right intracranial carotid close to the origin of the right middle cerebral artery.

Case 4 was the only example in this series of a patient who complained of "noises in the head and ears"; here a ruptured aneurysm was found on the left anterior cerebral artery at its junction with the

anterior communicating and a smaller unruptured aneurysm at the junction of the right carotid and posterior communicating arteries. In the literature of these cases buzzing noises in the head, hissing noises in the ears, waterfall noises, and even murmurs audible to the doctor, have been described.

In no single case in this series was any affection of the ninth, tenth, eleventh, or twelfth nerves below their nuclear origin discovered; and in no case did polyuria or glycosuria occur.

Pyrexia.—After a seizure consequent upon the rupture of the sac of a cerebral aneurysm of non-embolic origin, some degree of pyrexial disturbance is almost the rule, but in general the temperature reached is not high.

In Case 22 (p. 287) a rupture of an aneurysm on the posterior cerebellar branch of the basilar artery led before death to the development of a temperature of 105° F.; and in Case 18 the rupture of a similar aneurysm on the left anterior communicating artery to a temperature of 106° F.; but in no other case in this series did the thermometer record more than 103° F. In *five* cases (5, 6, 12, 13, and 29) the highest record was between 102° and 103° F.; in *two* cases (16 and 23) the highest point reached was 101° F., and in *six* cases (4, 14, 25, 27, 30, and 31) no temperature higher than 100° F. was observed, and in *ten* cases (2, 3, 9, 10, 15, 17, 21, 24, 26, 28) no record showing a higher figure than 99° F.

Stiffness of the neck, after the rupture of a basal cerebral vessel, is a not uncommon manifestation and source of complaint. The appearance of this symptom implies that blood has leaked into the posterior cranial fossa, and that clot has surrounded and compressed the lower medullary and upper cervical nerves. The presence of this symptom, therefore, renders probable the finding of the aneurysmal sac either on the basilar artery (or one of its branches) or on the posterior communicating artery. In general, however, the stiffness is both less troublesome to the patient and less persistent than it is in cases of basal meningitis. In Case 22 (p. 287) all the muscles of the back of the neck became painful and stiff, and the patient complained of severe occipital headache after the rupture of an aneurysm on the left posterior cerebellar branch of the basilar artery. In Case 29 (p. 292) a ruptured aneurysm on the left anterior cerebellar artery caused a similar complaint of stiffness of the neck, and made the patient hold his head flexed upon the left shoulder in the pose characteristic of a cerebellar lesion of the left side. In Case 30 (p. 249) the first symptom of which the patient complained was a left-sided

cervical stiffness, accompanied by intense pain and tenderness of the left occipital region. During the next twenty-four days these symptoms persisted. After death an aneurysm, which had leaked and allowed blood to escape into the basal cisterns, was found on the left posterior communicating artery near the bifurcation of the carotid.

At the present time, in cases of meningitis, the only certain signs of an inflammation of the membranes covering the cerebrospinal axis is the finding of organisms or of cellular exudate and products in the cerebrospinal fluid; just so, in cases of hæmorrhage into the brain and spinal cord, the only certain proof that some vessel has ruptured is the finding in the fluid removed by lumbar puncture of blood-cells and pigment more or less uniformly intermingled with the cerebrospinal fluid. Moreover, it is found that if the blood has escaped from a vessel in the cranial cavity, the longer the fluid after lumbar puncture is allowed to run, the more blood-stained the specimen becomes; whereas, if at the time of the insertion of the needle damage is done locally to a vessel in the lumbar sac, the first few cubic centimetres of fluid collected are found to contain the greatest amount of blood pigment and the greatest number of cells. If the hæmorrhage is of recent origin, the cerebrospinal fluid shows bright red staining and little altered red blood-cells; later the colour becomes paler and yellower, and the red cells more broken up.

In Case 5, on the day of the seizure, the cerebrospinal fluid obtained by lumbar puncture showed much bright red staining and many little altered red blood-cells; at autopsy this blood was found to have escaped from a burst aneurysm at the junction of the left anterior cerebral and anterior communicating arteries. On the day of death the cerebrospinal fluid of Case 29 (p. 292) was found to be under considerable pressure and to contain a few red cells, an excess of polynuclear cells, but no organisms, and to be stained slightly with recent, little altered, blood pigment. The blood in this case had escaped from an aneurysm on the left anterior inferior cerebellar artery. In Case 30 (p. 249) the cerebrospinal fluid was examined on three occasions—fifteen days, eighteen days, and twenty-four days after the first seizure; on each occasion the fluid was under considerable pressure, and contained much blood pigment and many red blood cells; and on each occasion it was noted that the later specimens obtained after lumbar puncture contained more pigment than did those first collected.

As a point in differential diagnosis, it is noteworthy that in cases of cerebral aneurysm the vessel whose wall gives way lies in the meninges

outside the parenchyma of the central nervous axis, whereas in cases of direct rupture or of rupture into a cerebral or cerebellar neoplasm the vessel gives way in the substance of the parenchyma, with the consequence that in aneurysm staining of the cerebrospinal fluid occurs at an earlier moment, and before the leakage is so profound. Moreover, as Wichern has noted, the finding of blood-stained cerebrospinal fluid is more likely to be of aid in early diagnosis if the aneurysm occurs on one of the vessels of the posterior cranial fossa.

Chapter IV.—The Clinical Manifestations associated with the Development of Embolic Intracranial Aneurysms.

Clinically, cases of intracranial aneurysm of embolic origin fall into a different nosological group from those intracranial aneurysms of congenital and degenerative origin. In the embolic class clinical evidence of a state of septicæmia and general infection is usually present when the patient first comes under observation, and in the great majority of cases, as Church [8] first pointed out in 1870, evidence of cardiac disease, of the ulcerative or progressive type, has preceded the onset of the cerebral manifestations. Between 1907 and 1913 at the London Hospital thirteen examples of embolic cerebral aneurysms occurred, and in these a history of several attacks of acute rheumatism followed by the development of a condition of progressive endocarditis was obtained in seven cases (Cases II, III, IV, VII, VIII, X and XII) and at autopsy pathological evidence of infected endocarditis was also found in Cases I, V and IX. Case VI was one of pyæmia subsequent to a retropharyngeal abscess, Case XI was one of pyæmia where no primary focus could be determined even at the *post-mortem* examination, and Case XIII one of puerperal septicæmia with general pyæmia.

In two cases only (Case I, p. 293, and Case IX, p. 294) did the patients first come under observation at hospital complaining of nervous manifestations and in both before death signs of valvular disease of the heart were discovered, and in both in addition a history was obtained of indefinite aches and pains preceding by some days the onset of the hemiplegic seizure.

Blood cultures were taken in Case III on three occasions with negative results; the blood in Case IV yielded a Gram-negative diplococcus, in Case V a short-chained streptococcus, in Cases VII and VIII a streptococcus and in Case XI a staphylococcus. In the other seven cases no blood cultures were made.

In Cases VI and XIII the patients were extremely ill, and although under observation as in-patients at hospital, at the time of death no local cerebral lesions were suspected. At the autopsy in Case VI a thick-walled unruptured cerebral aneurysm was discovered on the left anterior cerebral artery, and in Case XIII two ruptured aneurysms on small cortical vessels were found. Case XI before death fell into a severe typhoidal state, and although meningitis was suspected and thought to be consequent on a pyæmic abscess, no localizing nervous signs were discovered; in this patient the *post-mortem* examination revealed an aneurysmal dilatation and blockage at the bifurcation of the basilar artery.

Ruptures of embolic cerebral aneurysms leading to apoplectic attacks in patients who did not realize that they were ill occurred in Case I, a small girl of 7, whilst playing in the street; in Case VII, a man of 31, in the street when returning from work; and in Case IX, a girl of 15, whilst helping with house work.

Case I (p. 293) was admitted unconscious to the surgical side and gave the signs of a left hemiplegia: she was at first thought to be suffering from a "fractured base," but on the discovery of mitral disease a correct diagnosis was made before her death, which occurred six hours after the apoplectic insult.

Case VII was found lying in the street unable to speak and paralysed down the left side of the body; he was brought up to the hospital by the police, where eighteen days later he died. The first attack of acute rheumatism in this patient occurred sixteen years before death, and during the two and a half years before the cerebral infarction, breathlessness, headache, and joint pains had been extremely troublesome. At the autopsy a ruptured septic cerebral aneurysm with thick walls, of the size of a cherry, beneath the inferior extremity of the right ascending parietal convolution was discovered.

The clinical history of Case IX (p. 294) is most illuminating. She was admitted to hospital with a right hemiplegia and total aphasia, but showing no signs of cardiac disease. Lumbar puncture yielded sterile blood-stained cerebrospinal fluid, suggesting that some vessel supplying the central nervous system had leaked. As an in-patient the hemiplegic weakness largely disappeared and the power of speech returned, but twenty-two days after the seizure signs of mitral disease were recognized and she began to complain of abdominal pain. During the next week she vomited frequently, wasted and became very anæmic. No definite abdominal signs were ever discovered and she died thirty-two days after

the initial hemiplegic seizure apparently from exhaustion. At autopsy a ruptured aneurysm of the cœliac axis artery ruptured into the lesser peritoneal sac, a thrombosed aneurysmal dilatation of the left renal artery, a thrombosed aneurysmal dilatation at the bifurcation of the left intracranial carotid artery and progressive endocarditis without cardiac hypertrophy were discovered.

In Case X, the patient was at home in bed when he suffered his first seizure, and in Cases II, III, IV, V, VIII and XII the first nervous manifestations occurred whilst the patients were under observation and treatment in hospital for progressive endocarditis. Case II, a man, aged 41, with an extremely large heart and ever-changing signs of mitral and aortic disease, had been in hospital fourteen days, when he woke at 7 a.m. one morning feeling weak and faint; at 7.30 a.m. he suddenly fell back in bed complaining of weakness of the right arm, became comatose and died thirty hours later from a ruptured septic aneurysm on the right anterior cerebral artery. Case III, a boy, aged 16, had been in hospital nine and a half weeks suffering from increasing myocardial weakness and pyrexia, when he was suddenly seized with acute pain in the left side of his head. He immediately lost the power of speech and became hemiplegic on the right side. Consciousness was soon lost, and one and a half hours after the seizure with signs of respiratory failure he died. At the autopsy a septic aneurysm of the size of three peas, which had ruptured, was found on a large temporal branch of the left middle cerebral artery. Case IV, a young man, aged 22, had been in-patient three and a half months and had suffered from many attacks of pain due to the infarction of the organs, when fifty hours before death he suffered a slight generalized convulsion, became comatose and died. At autopsy an unruptured septic aneurysm at the anterior superior angle of the island of Reil on a branch of the right middle cerebral artery was found. Case V came into hospital complaining of cardiac disease and pain in the left upper thigh; eight days before death he was suddenly seized with intense pain above the left orbit. This pain lasted acutely for a few hours, and five days later, accompanied by transient blindness of left eye which lasted a few hours only, returned. Fifty-six hours before death he became distressed, vomited, lost consciousness and developed signs of a left-sided alternate hemiplegia, paralysis of the left third cranial nerve and paralysis of the right face, tongue, arm and leg. Two septic cerebral aneurysms were found at the autopsy, a ruptured one on the left anterior perforating artery and an unruptured one on a branch to the parietal lobe of the left middle cerebral.

Case VIII, a girl, aged 16, after a three months' stay in hospital, suddenly became comatose and died three days later from a ruptured aneurysm on the right middle cerebral artery, without ever recovering consciousness. Case XII, a man, aged 36, had been fourteen days in hospital, when for some hours in the evening he became restless and excited, and after the attack was found to have a dilated left pupil which reacted sluggishly to light; nine days later on waking about 6 a.m. he became loquacious, restless, and complained of a roaring noise in his head; two or three minutes later he lost consciousness and when seen at 8 a.m. was comatose and showed evidence of right-sided cortico-spinal interference. Thirty hours after this seizure he died, and at autopsy a large ruptured aneurysm, 2·5 cm. in diameter, on the left middle cerebral artery at the end of the horizontal limb of the Sylvian fissure, was discovered.

After the rupture of the aneurysm on the right middle cerebral artery in Case I a spastic left hemiparesis, convulsive movements of the right extremities and conjugate deviation of the eyes alternately to right and left were noted. In Case II an apoplectic seizure due to the rupture of an aneurysm on the right anterior cerebral artery led to a complete paralysis of the right arm and weakness of the right leg, with signs of bilateral pyramidal interference, and a fixed dilated pupil on the right side. Case III had suffered since early childhood from weakness of the left external rectus muscle and middle ear deafness on the left side. After the rupture of the aneurysm on a branch of the left middle cerebral artery speech was lost completely and the right face, arm, leg, and right upper eyelid became paralysed. In Case V, after the seizure due to the rupture of a septic aneurysm on the left anterior perforating artery, the left upper eyelid was completely paralysed, the power of speech was lost and paralysis of the right face, arm and leg was noted. Case VII was first seen twelve days after the seizure; at this time he could utter some words distinctly but could not propositionize and got angry when he could not answer simple questions of which, apparently, he knew the answer. He could copy simple written characters and diagrams and made attempts to draw common objects; he could not copy sentences or printed words. He complained of right parieto-occipital headache. The left arm and left leg were paralysed and the movements of the left face, left half of the palate and tongue were impaired. Signs of cortico-spinal interference to the left half of the body were present and sensibility on this half was altered. Gradually these signs improved and the patient became able to repeat

any simple combination of words. Four days before death he developed a nocturnal psychosis with excitement and next morning speech was again limited to a few words, which, though constantly uttered and misused, could not be repeated on demand. From this time on, his general condition got worse and for thirty-six hours before death he was comatose. The rupture had occurred near the temporal pole from a branch of the right middle cerebral artery. After a seizure Case IX became hemiplegic on the right side, showed paresis of the right face, became unable to put out her tongue, and developed proptosis of the left eyeball and optic neuritis on this side.

The first seizure in Case X occurred nine days before the patient came under observation and was followed by weakness of the left arm and some difficulty with speech. When admitted to hospital two days after a second seizure he was unconscious, and exhibited a hypertonic left arm, a spastic left leg, a flaccid right arm and a hypotonic right leg, paresis of the left face and evidence of bilateral pyramidal interference; the right pupil was dilated, the left of medium size: neither reacted to light. He died without ever recovering consciousness seven hours later. At the autopsy a partially thrombosed and ruptured septic aneurysm of the size of a cherry-stone was found on the right middle cerebral artery 5 cm. from its origin.

In the paper quoted above Sir John Rose Bradford [4] remarks: "My point is that a patient may die from cerebral aneurysm which may have resulted from embolism, but that at the time of death you may not be able to find any gross evidence of its embolic 'origin.'" If Case IX (p. 294) had died shortly after her admission to hospital, certainly progressive endocarditis would not have been diagnosed, for between January 15 and February 6, 1911, repeated examinations of the chest by a series of competent observers failed to reveal any abnormal cardiac signs; no murmurs were present, she was not short of breath and the extent of the projection of the heart against the chest wall was certainly not increased. After the latter date, however, unequivocal signs of mitral disease appeared. At autopsy there was endocarditis of both the mitral and aortic valves, but the size of the heart was not increased.

In the cases of cerebral aneurysms due to infected emboli, apart from the characters of the hemiplegias as signs of localizing value we had in Case IX unilateral (left-sided) proptosis and optic neuritis; the unruptured aneurysm was found at the bifurcation of the left intracranial carotid artery pressing upon the left cavernous sinus. Case V was a good example of the alternate hemiplegia known as the Weber syndrome,

left-sided ptosis and paralysis of the right face, tongue, arm and leg. Case VII showed a characteristic interference with internal speech and apraxia of the right upper extremity; a ruptured aneurysm was found near the temporal pole on the main branch of the right middle cerebral artery.

Many authors have laid stress upon the appearance of subjective noises in the head; in Case XII alone of this series is any mention made of this manifestation. This patient, before he lost consciousness, complained of an intense "roaring in the head"; here a large aneurysm 2·5 cm. in diameter was found at the end of the horizontal limb of the left Sylvian fissure.

Chapter V.—Summary of Conclusions.

(1) The clinical and pathological findings in 44 new cases of cerebral aneurysm are described.

(2) From the point of view both of the clinical history and the *post-mortem* appearances, the cases showing at necropsy aneurysmal formations on the basal cerebral arteries fall naturally into two groups, the non-inflammatory and the inflammatory.

(3) In a series of 5,432 consecutive examinations of the head cerebral aneurysms due to non-inflammatory changes in the middle wall of an artery occurred in 0·57 per cent., and aneurysms due to inflammatory changes in the cerebral arteries associated with infective embolism in 0·24 per cent.

(4) In the non-inflammatory group degenerative changes in the walls of the basal cerebral arteries were often found associated with high blood-pressure, cardiac hypertrophy and evidence of general arterial disease, but in many of the cases there was no evidence of excessive blood-pressure either at autopsy or from clinical observations, and the general arterial degeneration and the degeneration of the cerebral arteries was no greater than in the average subject of the same age.

(5) In the non-inflammatory group a congenital weakness of the arterial wall at junctional points is an important factor in their formation.

(6) The proportion of aneurysms associated with infective embolism in the cerebral arteries to those in all the other arteries of the body was as 15 to 9.

(7) In this series of cases no example of a cerebral aneurysm due to, or associated with, an infection by the *Spirochæta pallida* occurred, but 96·2 per cent. of the aneurysms of the aorta, 100 per cent. of the aneurysms of the other elastic arteries, and only 6·25 per cent. of the

aneurysms of the muscular and small elastic arteries, to which class the arteries of the circle of Willis and the cerebral arteries generally belong, were consequent on the local weakening of the arterial wall due to the inflammatory reaction set up by the activity of the *Spirochæta pallida*.

(8) Rupture of a cerebral aneurysm led to death in 0·44 per cent. of all cases of examination of the body, whilst direct rupture of an intracranial artery during the same period was the cause of death in 0·91 per cent.

(9) The largest aneurysm measured 30 mm. in diameter, but the great majority were much smaller.

(10) In 15 out of 31 cases (48·4 per cent.) of non-inflammatory cerebral aneurysms, whose ages varied from 53 to 19 years and averaged 38, no cardiovascular hypertrophy was present.

(11) The age-incidence of cerebral aneurysms of embolic origin is determined by that of progressive infective endocarditis and other chronic septicæmic and pyæmic conditions and is lower than in the non-inflammatory group.

(12) In 25 out of 31 cases (80·7 per cent.) of non-embolic aneurysm clinical manifestations attributable to cerebral hæmorrhage occurred; in 5 out of 31 (16·1 per cent.), no clinical manifestations of intracranial disease were recorded, and at autopsy an unsuspected and unruptured aneurysm was found; and in 1 case an unruptured aneurysm at the junction of the right carotid and middle cerebral arteries was found at the necropsy on a patient who for some time had suffered from frontal headache and had been deaf of his right ear.

(13) Amongst the cases which at autopsy showed ruptured non-embolic aneurysms two patients died before admission to hospital, and ten were admitted after a first apoplectic seizure and died without ever recovering consciousness.

(14) Multiple leakages of blood due to partial ruptures of the aneurysmal sac occurred in 13 out of 31 cases (41·9 per cent.). In these patients a history of multiple seizures of an apoplectic type occurred.

The clinical manifestations in this type of case form a definite nosological group rendering their diagnosis not difficult.

(15) The first rupture of the sac of a cerebral aneurysm was often brought about by a violent muscular effort or acute emotion.

(16) Before the aneurysmal sac ruptured signs of intracranial pressure were rare, but after this had taken place frequent; 10 cases out of 31 before death showed changes in the optic fundi.

(17) The headaches associated with non-embolic cerebral aneurysms have some localizing value.

(18) The interferences with intellectual and reflexomotor functions which occur in association with cerebral aneurysms, both before and after rupture, depend upon the site of the vascular deficiencies and the course which the hæmorrhage takes after rupture has occurred.

(19) The cranial nerves are frequently involved in blood-clot after rupture. Optic atrophy, bitemporal hemianopsia and lesions of the optic tract have been described, but the third pair of cranial nerves were those most commonly involved in this series of cases. Supraorbital neuralgia may be caused by pressure upon the Gasserian ganglion. In cases of aneurysms of the posterior fossa, and more especially of the cerebellar arteries, an involvement of the facial nerve after its exit from the pons was common. A subjective complaint of noises in the head occurred only in one case, and in this series no local affection in the posterior fossa of the ninth, tenth, eleventh, or twelth pair of nerves was noted.

(20) After rupture of aneurysms of the posterior fossa, a complaint of stiffness of the neck is a sign valuable in diagnosis.

(21) The finding of blood cells and blood pigment in the cerebrospinal fluid is the only clinical proof of cerebral hæmorrhage; in ruptured aneurysms staining of the cerebrospinal fluid occurs early.

(22) Of 13 cases of aneurysms on the cerebral arteries due to infective embolism, 10 were associated with ulcerative, progressive endocarditis, and in 5 cases a positive blood culture was obtained, but in 2 cases the onset of nervous manifestations first caused the patients to seek admission to hospital.

(23) Rupture of the sac of an embolic cerebral aneurysm leading to cerebral hæmorrhage and the death of the patient occurred in 9 out of 13 cases.

(24) The localizing signs in cases of embolic aneurysms are usually scanty.

LITERATURE.

[1] Bartholow. *Amer. Journ. Med. Sci.*, 1872, vol. lxiv, p. 373.
[2] Beadles. *Brain*, 1907, vol. xxx, p. 285.
[3] Bradford. *Brit. Med. Journ.*, 1894, vol. ii, p. 869; *Trans. Clin. Soc.*, 1895, vol. xxxviii, p. 1.
[4] *Idem*. *Lancet*, 1908, vol. ii, p. 703.
[5] Brinton. *Trans. Path. Soc.*, 1852, vol. iii, p. 49.
[6] Bruns. "Die Geschwülste des Nervensystems," 1908, ii Aufl., S. 59.
[7] Byrom Bramwell. "Clinical Studies," July 2, 1906, p. 289; *Rev. of Neurol. and Psychiat.*, 1906, vol. iv, p. 507.

[8] CHURCH. *St. Bartholomew's Hosp. Reports*, 1870, vol. vi, p. 99.
[9] EPPINGER. *Arch. f. klin. Chirur.*, 1887, vol. xxxv, Supplem. S. 1.
[10] GOWERS. "Diseases of the Nervous System," vol. ii, second edition, 1893, p. 529.
[11] GULL. *Guy's Hosp. Reports*, 1859, vol. v, p. 281; New Sydenham Society's Publications, 1894, vol. cxlvii, p. 86.
[12] HALE WHITE. *Trans. Clin. Soc.*, 1895, vol. xxxviii, p. 1.
[13] HOFMANN. *Zeitschr. f. Morphol. und Anthropol.*, 1900, vol. ii, S. 247.
[14] LANGBEIN. *Münch. med. Wochenschr.*, 1913, vol. lx, S. 22.
[15] LEBERT. *Berlin. klin. Wochenschr.*, 1866, vol. iii, S. 60, 209, 229, 249, 282, 336, 345, 387, 402.
[16] NEWTON PITT. *Brit. Med. Journ.*, 1890, vol. i, p. 827.
[17] OSLER. "Principles and Practice of Medicine," eighth edition, 1912, p. 982.
[18] OSLER and MACRAE'S "System of Medicine," 1908, vol. iv, p. 459.
[19] PARNISETTI. Quoted by Rothmann.
[20] PEACOCK. *St. Thomas's Hosp. Reports*, 1876, new series, vol. vii, pp. 119 and 317.
[21] PONFICK. *Virchow's Arch. f. Path., Anat. und Physiol.*, 1873, vol. lviii, S. 528.
[22] ROTHMANN. *Arch. f. Psychiat.*, 1904, vol. xxxviii, S. 278.
[23] SIMMONDS. *Deutsch. med. Wochenschr.*, 1901, vol. xxvii, S. 353.
[24] TURNBULL. *Quart. Journ. Med.*, 1915, vol. viii, p. 201.
[25] VON HOFMANN. *Wien. klin. Wochenschr.*, 1894, vol. vii, S. 823, 848, 867, 886.
[26] WEIR MITCHELL. *Journ. Nerv. and Ment. Dis.*, 1889, vol. xvi, p. 44.
[27] WICHERN. *Münch. med. Wochenschr.*, 1911, vol. lviii, Nr. 51, S. 2224.
[28] *Idem. Deutsch. Zeitschr. f. Nervenheilk.*, 1912, vol. xliv, S. 220.
[29] WINDLE. *Journ. Anat. and Physiol.*, 1888, vol. xxii, p. 289.
[30] WYRUBOW. Quoted by Rothmann.

APPENDIX.

(A) NON-EMBOLIC ANEURYSMS.

A case in which death occurred from the rupture of an aneurysm at the junction of the right anterior cerebral and anterior communicating arteries in a man aged 41, *who had been under more or less continuous observation at hospital over a period of sixteen years, and had complained of recurring attacks of headache and been blind of an anæsthetic left eyeball for three years. The first seizure due to the rupture of the sac occurred forty-three days, a second twenty-five days, a third seven days, and a fourth twenty-six hours before death in coma.*

Case 23 (32377/1903, 30306/1906, 30181/1909).—Edward C., aged 41, grocer, single, was admitted to the London Hospital under the care of Dr. Cecil Wall on January 21, 1909, and died on January 25. He had been under observation and in-patient at various times during the preceding twelve and a half years. In 1893, at the age of 25, he attended as an out-patient and was treated for "colic and left wrist-drop" with aperients, electricity, and liniments. At this time the case was diagnosed as one of "(?) plumbism"; under treatment his symptoms disappeared. In 1896 he was readmitted suffering from chronic diarrhœa; after seven weeks' treatment he was discharged relieved. From this time until his death he was subject to episodical attacks of diarrhœa and melæna. In 1903 enteroptosis was diagnosed, and he was

treated with massage. In the full histories taken on these admissions no mention is made of any headache or abnormality of the left eye. Between 1902 and 1903 he suffered two attacks of hæmatemesis, and during the years 1896 to 1903 his weight varied greatly, between 11 st. 7 lb. and 8 st. 4 lb.; he stated that when his weight was low he was subject to attacks of diarrhœa and left-sided abdominal pain.

In January, 1906, he began to complain of headache, sleeplessness, and a feeling of confusion immediately after waking, in addition to his old symptoms of flatulence, attacks of diarrhœa alternating with periods of obstinate constipation and left-sided abdominal pain. A note made at this time states that he was blind of the left eye, following iridectomy for staphyloma, but does not give the date of the operation, and further states that the cornea of the shrivelled eyeball showed scars of old corneal ulcers. At this time no abnormal nervous signs were discovered; the heart was not enlarged and the cardiac sounds were clear. An explanation of his symptomatology was found in the profound enteroptosis and the finding of a right movable kidney and occasional albuminuria. The stomach was greatly dilated and prolapsed; when the patient stood erect the small intestines fell forwards and the hypogastrium bulged; the transverse colon could be moved vertically over a distance of 3 or 4 in. Under massage and hospital treatment his weight in sixteen days rose from 8 st. 4 lb. to 9 st. 1 lb. On discharge he was given an abdominal belt, and three months later reported himself as "free from all the old symptoms except the headache."

On December 12, 1908, forty-three days before death, he was seized with a "bad influenza"—dreadful headaches day and night, pains in the back of the head and neck, constipation, and vomiting. He remained in bed four days. During these days he retched frequently, his motions were loose and he complained of severe frontal and vertical headache. For the next two weeks he was constantly ailing; the headache never ceased, but he was able to attend to his shop. On December 31, 1908, twenty-five days before death, in the morning about 4.30 a.m., the headache became "worse than ever," he felt "really bad in himself," got out of bed and "started cursing everybody, using very foul language." After this attack until death he did no more work and complained of headache "always present but varying in intensity from day to day and hour to hour." On January 17, 1909, he suffered another exacerbation in symptoms. The pain in the head on this day seemed to begin in the back of the head just beneath the hairy region and shoot forward to between the eyes and "almost push my eyes forward." On the 19th he vomited several times quite apart from the intake of food, which he usually refused, and became extremely constipated. On the night of January 20 to 21 he became very excited, cried out for a knife with which to murder his wife, could not be kept in bed, and beat his children. He was given injections of morphia, and next morning admitted to hospital.

On admission.—The patient was a thin, anæmic man weighing 7 st. 8 lb He was extremely irritable, resenting all interferences and exposure to light.

At times he was noisy and called for objects with which to commit murder on his relations. In a general way he was intelligent, talked freely and answered questions lucidly. He could not give any connected account of his actions and mode of life during the preceding weeks, but readily answered questions relating to his shop and previous admissions to hospital. He complained of much severe left-sided frontal headache and said that the pain was increased by pressure between the eyes. After admission he vomited frequently; the vomits were small, bile-stained and consisted almost entirely of altered and clotted milk; the vomitings bore no relation to the intake of food.

Speech was unaffected and attention feeble, his remarks flitting rapidly from thought to thought. There was no paralysis or weakness of any extremity. The grasps were equal and powerful. He complained of no spontaneous pains in the limbs and to testing no loss of tactile, painful or thermal sensibility was discovered. The answers to tests for the recognition of posture and passive movement were poor and defective compared with those obtained in other modes of examination. No trophic or vasomotor changes were noted.

The knee-jerks were glib and equal on the two sides. Ankle-clonus was not obtained. No plantar responses were obtained. The arm-jerks were natural.

The left eyeball was small and atrophic, and with this eye the patient could not appreciate light. The left fundus could not be seen; the right appeared healthy, and the edges of this disc were clearly seen and sharply defined.

There was definite ptosis of the right eye; this eyeball moved badly but no local weakness of any single muscle could be determined. Nystagmus was not present. The right pupil was small, regular in outline, and reacted normally to light and convergence. The movements of the face, jaw, tongue, palate and larynx were unaffected.

The patient suffered from retention of urine, and catheterization was necessary: the bowels were only opened after the administration of enemata.

The tongue was dry and the pharynx filled with dried mucus. The chest was long, flat, phthinoid, the intercostal spaces wide and oblique, the sub-costal angle narrow, and the costal margins everted; a Harrison's sulcus was present and the sternum was depressed. The neck was thin and the trapezii long and sloping. The apex-beat of the heart was felt in the fifth interspace in the mid-clavicular line. No murmurs were heard. There was no abnormal dulness over the lungs and the breath sounds were natural.

The abdominal wall was thin and the distance between umbilicus and pubes long; the outlines of the stomach which filled the epigastric and upper umbilical region could be seen, and splashing was easily elicited. The liver and spleen were not enlarged. The right kidney was easily felt and could be seized between the fingers. The urine contained a trace of albumin, but no sugar.

On the morning of January 24 the patient suddenly cried out, "Lift me up, it's coming again." A nurse rushed up to him and lifted him up: then he began to jerk his arms, head and legs. He did not bite his tongue. His

colour changed and the pulse became feeble and rapid. After about half a minute the movements became more violent, then general rigidity followed by a state of deep unconsciousness and snoring breathing set in. He remained stiff for some minutes and gradually fell into a state of heavy sleep. After the seizure he was found to have passed urine and fæces under him. During the next twenty hours he occasionally raised his right arm and waved his right hand or picked with it at the bedclothes. No absolute paralysis appeared, but the left arm became more flaccid than the right. On January 25, twenty-six hours after this seizure, he died of respiratory failure.

At the *post-mortem* examination made on January 26 (P.M. 87/1909), a ruptured aneurysm of the size of a currant-grape was found at the junction of the right anterior cerebral and anterior communicating arteries, from which blood had escaped, covered the frontal lobes, filled the superior longitudinal fissure and the basal cisterns, and had spread over the whole of the cerebellum. The frontal lobes were stained brown. The substance of the hemisphere around the aneurysm was softened and had allowed blood to escape into the lateral, and thence into the third and fourth ventricles. The brain weighed 1,545 grm. The left optic nerve and left eyeball were atrophic. In the aneurysm itself and in the other cerebral arteries no evidence of atheroma was obtained. A few small patches of atheroma were found in the coronaries, and in the aortic commissure and abdominal aorta there were one or two small plaques and in the femorals slight atheromatous changes, but the radials were neither thickened nor tortuous. The heart was not hypertrophied but the kidneys showed fine granular changes and a few cysts. The lungs, liver and kidneys were greatly congested. The contents of the small and large intestines were blood-stained. This blood had escaped from a sub-mucosal point in the first part of the duodenum.

A case of aneurysm of the left posterior cerebellar branch of the basilar artery which at autopsy was found to have ruptured and given rise to a mass of blood-clot which compressed the pons, cerebellum, medulla, and the occipital lobe of the left cerebral hemisphere in a married woman, aged 42, who, when aged 25, and eight months pregnant of her second child, had suffered a transient hemiplegia affecting the right face, tongue, arm and leg, and who for five months before death had complained of slight epileptiform (petit mal) attacks with visual sensations. Nineteen days before death this patient had a seizure, followed by a second twelve days and a third ten days before the fatal issue. On admission to hospital, delirium, paresis left face and right hemiplegia of arm and leg, tenderness in left occipital region and stiffness of the upper part of the neck, left cerebellar posture of head, no nystagmus, early optic neuritic changes right.

Case 22 (43124/1909).—Anne S., aged 42, married, was admitted to the London Hospital under the care of Dr. F. J. Smith, on November 19, 1909, and died on November 24. She married in 1885, and had had two children and no miscarriages. No history suggesting primary or secondary syphilis

could be obtained. In 1889, at the age of 22, left inguinal colotomy for "stricture and ulceration" of the rectum was performed at St. Mark's Hospital. This stricture was thought to have had syphilitic origin. At the age of 25, when eight months pregnant of her second child, she suddenly woke one morning to find the right half of her body—face, tongue, arm and leg—paralysed and some difficulty with speech. The face and speech rapidly improved, and before her baby, which is still alive and now a healthy young woman, was born, the weakness of the extremities had largely disappeared. During the years 1892 to 1908 her health was good.

Between June and November, 1909, the patient had suffered five or six attacks of "blindness lasting a few minutes." In these attacks she never lost consciousness, and after ten minutes or so seemed perfectly well. The attacks were not accompanied by any headache, and in them she never vomited.

On November 5, 1909, nineteen days before death, she was seized with a sudden severe pain in the head and a sensation as of a "pulling of the back of her head as if her head were in a vice." This severe pain in the occipital region was accompanied by "aches all over." After the attack the left half of her face became "funny and weak." On November 12, she suffered from another "turn" in which she vomited once and became very "queer" and "quiet." On November 14, she suffered a second seizure "exactly like that on the 5th." The headache at this time was severe and her neck became "fearfully stiff and tender." After the headache ceased she was drowsy and suffered from "fever." With these attacks no weakness of the extremities was noticed.

On admission, the patient was an obese woman, weighing 13 st. 2 lb., who looked old for her years. She lay in bed moaning and groaning, and absolutely helpless. Shortly after admission delirium with outbursts of loud, frequent shouts of foul and filthy language set in. She mistook the people around her for her own relatives, and abused them. She was suspicious, but at times would listen to reason, and could with persuasion be got to answer questions. On command, simple orders were carried out. She complained of pain and tenderness over the region of the left occiput and nape of the neck, and greatly resented all attempts at turning her head. The head was held strongly flexed with the chin pointing towards the left. Under observation, she did not vomit, nor did she exhibit any irregular, involuntary movements.

The right arm and leg were spastic and paretic. All movements on this half of the body were slowly carried out and clumsily performed. No sensory loss was discovered. The right knee-jerk was exaggerated, the left could not be obtained. Ankle-clonus was not elicited. On the right side the plantar response was extensor, on the left flexor. The arm-jerks on the right side were brisk, on the left only doubtfully obtained.

The left fundus appeared healthy. On the right side the veins were engorged, the arteries unaffected, and the edges of the pink disc indistinct and blurred. Hearing was not affected.

Ocular movements were natural. Ptosis and nystagmus were absent. The

pupils were small, regular in outline, and reacted definitely, though sluggishly, to light. The left face was paretic in its lower half, but moved fairly in emotion. The tongue could be protruded straight and held steadily. The sphincters were under control.

The rate of the pulse varied between 96 and 120. The temperature was raised to 103° F. Respirations were shallow—36 per minute. The tongue was thickly furred. The chest moved naturally. The heart was not enlarged and the cardiac sounds were natural. In the left inguinal region was an old colotomy wound, with healthy appearance. The liver reached below the costal margin, and its edges could just be felt. The urine contained neither albumin nor sugar.

On the nights of the 20th and 21st the patient became wildly delirious and excited. Next morning she was comatose, and the right side of the face was seen to be paretic. After this the pulse began to fail, and became variable in force and rate, tending to about 88 per minute. On the 22nd she could be roused with difficulty, cried out repeatedly, understood apparently some commands, and at times uttered a feeble unintelligible reply. Death occurred with increasing coma, grouped rhythmic respirations, flushing and cyanosis of the face and hyperpyrexia (T. = 105·6° F.) on the morning of the 24th.

At the autopsy (P.M. 1166/1909), performed on November 25, a ruptured aneurysm on the left posterior cerebellar branch of the basilar artery was found. The middle peduncles of the cerebellum, the pons, and the medulla oblongata were compressed on the left side by a lump of firm, brown-pigmented clot, and the meninges around this mass of clot were deeply stained, and of a red-brown colour. There was no softening of the brain, which weighed 1,134 grm. No evidence of atheroma was found either in the aneurysm or in the other cerebral arteries, but in the arteries of the general circulation moderate atheromatous changes were seen as fatty and calcareous plaques scattered here and there over the aorta, coronaries, &c. The heart weighed 361 grm. The lungs showed severe acid digestion and acute purulent bronchitis of the lower lobes. Two and a half inches above the anal orifice was an impervious stricture of the rectum, for the relief of which a permanent colostomy had been made; below this opening the mucous membrane of the colon and rectum was contracted and atrophied.

A case in which the partial rupture of an aneurysm at the junction of the left anterior cerebral and anterior communicating arteries occurred suddenly in man, aged 53, whilst watching a football match. Attacks of drowsiness and delirium, epileptiform seizures, clumsiness in movements, left frontal headache, left optic neuritis. Death on twenty-seventh day.

Case 16.—30191/1911: Joseph M., aged 53, married, was admitted to the London Hospital under the care of Dr. Francis Warner on January 26, and died on February 4, 1911. He had followed the trade of carpenter for thirty-five years, and until the onset of the illness from which he died

had enjoyed good health. He married at the age of 26, was a widower, and by his wife had had five children, four of whom survived, aged respectively 25, 23, 21, and 19 years. He denied venereal infection and exposure. In early life he had had attacks of "typhoid" and "typhus" fevers; in recent years he had suffered on three occasions from "influenzal colds with fever and pains in the back."

During the latter part of December, 1910, and the first days of January, 1911, he had been feeling particularly well. On January 7 he went to watch a football match, and, whilst looking on, "came over queer, shivered, and developed severe frontal headache." He was taken home in a cab and put to bed. Consciousness was not lost; vomiting set in some hours later on the same day; he vomited several times, and brought up large quantities on each occasion. With the headache and vomiting he himself became irritable and ill-tempered. The pains in the head were chiefly vertical and frontal. He remained in bed at home from the 7th until the time of admission, complaining of a persistent headache of varying intensity; whilst in bed at home he vomited on several occasions, and was very constipated. Suddenly on the night of the 12th he became noisy, talkative, and delirious, saw black objects before his eyes, and made attempts as if to seize them. Next morning he was better again, and seemed "his normal self." A few days later, however, he began to "talk strange, and behave as if not right in his mind," but at the same time slept well. On January 19 he suffered from his first "fit"; in it he threw himself about, opened and shut his mouth repeatedly, and turned his eyes upwards. On the nights of the 20th and 21st he had three such attacks, "lasting on him altogether two hours and a half." On admission his daughter said that he had not recognized anybody since the 22nd, and had passed his urine and motions into the bed since the 24th. A letter from his doctor stated that albumen had been present in the urine on the 7th.

On admission, the patient was a well-preserved man, aged 53, of healthy appearance. He was dazed, drowsy, and lethargic. In answer to questions, he usually gave some reply after a pause, but the reply was pointless and irrelevant, rather than incorrect. He could give no connected account of himself, and when asked who he was, where he lived, or how long he had been ill, merely uttered some incomprehensible sounds. Apparently hearing was not affected. Attention after he had been roused was fleeting. He took no interest in his surroundings, and in general lay vegetable-like. Spontaneous movements of the hands were occasionally seen, but of the face and legs rarely. He showed evidence of recent wasting, was dusky and cyanosed; his face was expressionless and drawn. The mouth usually remained half-opened, exposing an extremely harsh brown tongue. The lips were dry and the pharynx filled with mucus. The fairly preserved teeth were covered with sordes and dried secretion. The skin was harsh and dry, and œdema was not present. Large varicose veins coursed over both lower extremities, and pigmentary scars, apparently secondary to old and healed varicose ulcers, were seen on the front of the shins. No suggestion of syphilis was discovered.

The apex-beat of the heart was felt in the fifth interspace, a quarter of an inch outside the mid-clavicular line. The area of superficial cardiac dulness was small. The clavicles were raised, the sternum fixed, the chest barrelled, and the respiratory movements small. No abnormal dulness was found over the lungs. The breath sounds were feeble and distant, and expiration was prolonged. No abnormal cardiac bruits were heard. The rate of the pulse was 96 per minute; before death it fell to 66. The temperature was slightly raised, to 99·5° or 101° F. throughout the illness. The superficial arteries were tortuous and felt thickened. The blood-pressure, as measured by the Riva-Rocci apparatus in the right brachial artery, was 180 mm.

The liver was not enlarged and its edge could not be felt. The splenic dulness was not large and the lower pole was not palpable. No abnormal signs were found in the abdomen. The urine contained albumin and a thick cloud was obtained on boiling. The specific gravity varied from 1028 to 1030. On standing a thick deposit of amorphous urates was obtained. Sugar was not present.

On the three days preceding death, February 1, 2, and 3, the patient exhibited several epileptiform attacks: the movements chiefly affected the left upper extremity and were followed by loss of consciousness, dilatation of the pupils, and the disappearance of the corneal reflex. Each attack lasted from a half to two minutes and was accompanied by relaxation of the sphincters. After the attacks he sank into a quiet and drowsy state. Headache was a constant complaint and was referred to the left temple and above the left orbit. As an in-patient he did not vomit.

There was no absolute paralysis of any limb. Irregular, inco-ordinate, clumsy movements of both hands occurred from time to time. Spasticity was not present and the tone of the muscles was little altered. Sensation was difficult to test, but no loss could be determined. Dermatographia was easily elicited.

The knee-jerks were exaggerated, the response on the right side was somewhat brisker than on the left. Ankle-clonus was not obtained. The arm-jerks were brisk. On both sides the plantar reflexes gave a flexor response. The abdominal and cremasteric reflexes were just obtained. The left corneal reflex was readily elicited, the right only doubtfully obtained.

The right optic disc and fundus appeared pale, the veins were full, but the arteries small and the edges of the disc sharp. On the left side the disc was swollen and its edges blurred; the veins on this side were larger than on the right but the arteries appeared fairly normal. The drums of the ears appeared healthy.

Ptosis and nystagmus were absent and ocular movements free. The pupils were equal and reacted sluggishly to light. Facial movements were rarely seen, but no paralysis could be made out. The movements of the tongue, larynx and palate were unaffected.

The patient suffered from retention of urine and dribbling overflow incontinence. He was constipated and the bowels were only opened with

enemata. Death occurred from respiratory failure on February 4, twenty-seven days after the first seizure.

At the autopsy (P.M. 104/1911) performed on February 4, a ruptured cerebral aneurysm, of the size of a pea, was found at the junction of the left anterior cerebral with the communicating arteries. Around the aneurysm was much hæmorrhagic matting of the meninges. All the cerebral ventricles were filled with blood-clot which had found an entrance through into the left lateral ventricle by local softening and erosion. At the base of the brain was much intradural and intraarachnoid hæmorrhage. The brain weighed 1,535 grm. There was no visible atheroma of the cerebral arteries, but slight atheroma was seen elsewhere, a few fat-flecks in the coronaries, and pin-head fat-flecks in the commissure and descending aorta. The weight of the heart was 290 grm. The lungs were emphysematous and showed a fibro-calcareous nodule at the apex of the left lung and a few smaller nodules in the right upper lobe. The other organs appeared healthy.

On microscopic examination the aneurysm was found to have a thin wall made up of fibrous tissue with some slight fusiform thickenings. Some of these thickenings appeared to be fibrosed muscularis, others fibrotic thickenings of the intima in which a few elastic fibres were seen.

A case in which the rupture of an aneurysm of the left anterior cerebellar artery led six days later to the death of a young man, aged 22. Drowsiness, left facial paralysis, bilateral corticospinal interference, left cerebellar posture of head, stiffness of neck and blood in the cerebrospinal fluid.

Case 29 (32669/1909).—Arthur P., aged 22, stable-boy, married, was admitted to the London Hospital, under the care of Dr. Leyton, on November 24, 1909, and died on November 25.

His father stated that his health had been good until November 19, 1909. In 1903 he had been laid up in bed at home for six weeks with "rheumatic fever," and in 1907 he suffered an attack of "gonorrhœa." On November 19, while in the stable he fainted, but after the faint did a full day's work, going a complete round with the cart. On the 20th he refused his food, said he "felt queer" but made no specific complaints. On the 21st he got up but was "half-asleep, talked to himself, and would not listen to what was said." On the 22nd and on the morning of the 23rd he seemed better, but about 11 a.m. on the latter day he had a seizure. At the time he was walking about the house, when suddenly he reeled, came over stupid and deaf. He was put to bed and when found at 5 p.m. was deeply unconscious. A doctor was called and shortly after midnight he was admitted to hospital.

On admission, he was semi-conscious and in general lay quiet in bed on his left side. He resented all interference and when spoken to made some unintelligible replies, continuing thereafter to mumble to himself. His face was flushed, his breathing stertorous. On the bridge of the nose was a scar obtained in his fall on the 19th. The right side of the heart was dilated but there was

no evidence of any enlargement of the left ventricle. At the apex a systolic murmur was audible. With the exception of a few rhonchi, no abnormal signs were discovered over the lungs. The urine contained a trace of albumen but no sugar and no casts. The temperature varied between 101° and 102° F., the rate of the pulse from 92 to 116 and of respirations from 32 to 38 per minute. The blood-pressure in the brachial artery measured 110 mm.

The optic discs and fundi appeared healthy. All the ocular movements and the movements of the pupils were unaffected, but the patient could not be persuaded to fix his gaze upon any object. The left side of the face both in its upper and lower halves was paralysed. The tongue could not be protruded.

Both arms were held stiffly, the right more so than the left. The left arm from time to time twitched in an irregular manner. The right leg was paretic and the tone in its muscles increased. There was no rigidity of the muscles of the neck but the head was held flexed towards the left shoulder and the chin pointed towards the right. Sensation could not be tested.

The knee-jerks on both sides were exaggerated, ankle-clonus was elicited from both ankles, and the response from the plantar reflex on each side was extensor. Both wrist- and elbow-jerks were brisk. The abdominal reflexes could not be obtained. Urine and fæces were passed incontinently.

Consciousness was never regained. On the 25th the eye-movements were more dissociated and irregular and the head more definitely rotated to the left; true nystagmus, however, was not obtained. Intense photophobia was present. The reflexes varied, at times ankle-clonus was readily obtained, at other times this sign was absent. On the 25th lumbar puncture yielded clear fluid under pressure which contained a few red cells, excess of polynuclear cells, but no organisms. Death occurred six days after the initial "faint" on November 25 from respiratory failure.

At the autopsy (P.M. 1166/1909), a ruptured aneurysm of the size of a pea was found on the left anterior inferior cerebellar artery. All the other cerebral vessels appeared healthy, there was no cardio-vascular hypertrophy and all the organs except the lungs, which showed broncho-pneumonic changes, appeared healthy.

(B) Embolic Aneurysms.

A case of infected endocarditis in a child aged 7 years, in whom, after a period of slight malaise, death occurred from cerebral hæmorrhage seven hours after a seizure consequent upon the rupture of an embolic aneurysm on the right middle cerebral artery.

Case I.—20861/08, Bessie G., aged 7, was admitted to the London Hospital under the care of Mr. Percy Dean on April 7, 1908, and died seven hours after admission.

The mother stated that the child had been ailing for some days but had never been really ill until the day of admission. On this day, whilst playing on the stairs, she suddenly fell down four steps and struck her forehead against

the stairs. Immediately after the accident she was brought up to the hospital and admitted.

The patient was a well-developed child of 7. On admission she was unconscious. At intervals she showed irregular movements of the right upper and lower extremities; in these attacks the eyes were observed to roll slowly outwards, first to the left and then to the right, and the mouth was seen to twitch in an irregular fashion. The left upper and lower extremities were paralysed and stiff.

Both knee-jerks were brisk. On the left side the plantar reflex gave an extensor response, on the right a flexor.

The area of cardiac dulness was increased to the right of the sternum, but the apex-beat was not displaced either outwards or downwards. At the apex the first cardiac sound was replaced by a long, blowing, systolic murmur which was well heard in the axilla. The pulmonary second sound was accentuated. No abnormal physical signs were discovered in the lungs and no signs of any injury were present. The rate of the pulse was 68 per minute and of respiration 27 per minute, and the temperature was 96° F.

Chloral was administered to the patient by the mouth, but five hours later respiration began to fail. Artificial respiration was carried on for half an hour, at the end of which time the pulse could no longer be felt at the wrist.

At the autopsy performed on April 8, 1908 (P.M. 345/1908), a ruptured aneurysm of the size of two peas was found on the right middle cerebral artery about 1½ in. from its origin. The posterior part of the basal ganglia and the internal capsule had been almost completely destroyed and blood-clot was found in both lateral and in the third and fourth ventricles, whilst the convexity of the right cerebral hemisphere was covered with sub-arachnoid hæmorrhage. The heart, which weighed 113 grm., showed the appearances of progressive endocarditis, with large fibrinous vegetations on the mitral and aortic valves. The spleen was septic and the organs engorged. No other aneurysms or infarcts were discovered. Microscopic examination showed that the aneurysm was filled with organizing blood-clot and that the aneurysmal wall exhibited an area of necrosis with much round-celled and purulent infiltration around the artery.

A case of infected endocarditis with multiple infarctions and the formation of embolic aneurysmal dilatations on the left intracranial carotid, the left renal and the cœliac axis arteries in a girl aged 15. *Onset of acute manifestations with a seizure followed by aphasia and right hemiplegia thirty-two days before death: cardiac affection first recognized seven days before death, which occurred in consequence of bleeding into the lesser sac of the peritoneum.*

Case IX.—40136/1911, R.H.—Mary N., aged 15, was admitted to the London Hospital under the care of Dr. Robert Hutchison, on January 15, 1911, and died on February 15.

As a baby the patient had suffered only from attacks of whooping-cough

and measles. She had never suffered from acute rheumatism or scarlet fever and had had good health until the onset of the illness for which she was admitted to hospital. About four weeks before admission, her mother stated, she had had a blow upon the head. This blow was not followed by untoward symptoms, but from it her mother dated the present illness. On January 8, 1911, the patient began to complain of indefinite aches and pains in her arms, legs and back; they were, however, slight, and she was not confined to bed. On January 14, the day before her admission, she had a seizure. She fell down, clenched her teeth and voided urine and fæces. The seizure was followed by a period of restlessness marked chiefly by irregular movements of the left arm and left leg. After the seizure until the time of her admission she never spoke.

The patient was a fairly well developed Hebrew girl of 15, with dark hair. She was not anæmic and the lymphatic glands were not enlarged. She was drowsy, irritable, and only moved when stimulated or in response to loud noises. No wounds were discovered on her head or body. The cheeks were flushed, more especially on the side of the face on which she had been lying. Erythematous patches were seen at all points of pressure.

The right arm and right leg were paralysed; the right arm was stiff, the right leg flaccid. High-grade movements of a choreio-athetotic type of the left hand occurred from time to time. The left leg was moved freely, apparently under volition.

The right knee-jerk was exaggerated and knee-clonus was obtained on this side; on the left side the knee-jerk was readily obtained, but was not exaggerated. No ankle-clonus could be obtained. On the right side the plantar reflex gave an extensor response, on the left a flexor. On the right side no abdominal reflexes could be obtained, on the left they were brisk. The elbow-jerk and wrist-jerk on the left side were normal, on the right these could not be elicited. The jaw-jerk was normal.

Sensation could not be tested. The right arm was blue and cold, the left of normal appearance.

The right optic disc appeared healthy, but the edges of the left were blurred and the whole fundus on this side had a ground-glass appearance. The drums of both ears appeared healthy.

Ocular movements were well carried out. The right eyeball appeared more sunken than the left. The pupils were equal, of moderate size and reacted briskly to light. The lower half of the right side of the face and the levator palpebraris on this side were paretic. The tongue could not be protruded. The head was rotated slightly to the left and the sternomastoid muscle on the right side appeared to be completely paralysed.

The patient was constipated and the bowels only moved after the administration of enemata. Urine was passed incontinently.

On admission the rate of the pulse was 120 per minute; the beats were regular and equal. The apex-beat of the heart was seen and felt in the fifth space just inside the mid-clavicular line. At all the areas both cardiac sounds

were heard and no murmurs were present. The pulmonary second sound was slightly accentuated. The rate of respiration was 24 per minute. The movements of the chest were unaffected and the breath sounds were unaltered. The abdomen was somewhat sunken. Neither the liver nor the spleen could be felt, and the urine contained neither albumen nor sugar. The lips were dry and the tongue coated with a brownish fur. The tonsils were large.

The temperature on admission was 100° F. The temperature for eight days after admission was raised and somewhat irregularly hectic, ranging between 101° in the evenings and 99° in the mornings. On the three days before death after an apyrexial period, the evening temperature rose, and on the evening of February 13 reached 102° F.

On January 16 lumbar puncture was performed and 30 c.c. of cerebrospinal fluid were withdrawn. This fluid contained many red blood corpuscles, a few lymphocytes and a large excess of polynuclear cells. On January 26 lumbar puncture was again performed; the fluid on this date was under pressure, but on centrifugalization it yielded no cells and cultures made from it proved sterile.

Ten days after admission the patient began to talk again, and the right hemiplegic weakness commenced to clear, and until about February 6 the patient seemed to be making a good recovery; on this day the patient vomited and the first cardiac sound became much weaker. On February 8 a loud mitral systolic murmur was heard for the first time. After this date the patient became more ill, wasted, refused food and gradually sank, apparently from exhaustion and increasing anæmia. Death occurred on February 15, thirty-two days after the initial seizure.

At the autopsy performed on February 15 (P.M. 137/1911), an aneurysm, 3 cm. in diameter, partially walled with blood-clot, was found on the cœliac axis artery. This aneurysm had ruptured into the lesser peritoneal sac. The peritoneal cavity contained 595 grm. of blood-clot and 1,136 c.c. of serous fluid. The heart, which weighed 141 grm., showed a condition of ulcerative endocarditis, there being one large slimy, polypoid and many small granular vegetations on the mitral valve. In the hilum of the left kidney the left renal artery was found dilated and thrombosed; this thrombosis had given rise to a total infarction of this kidney. The right kidney showed a condition of acute hæmorrhagic nephritis. The spleen was septic and weighed 169 grm., and the liver showed much parenchymatous degeneration.

At the point of bifurcation of the left intracranial carotid artery was a thrombosed aneurysmal dilatation caused by a septic embolus. The left basal nuclei and the adjacent part of the left centrum semi-ovale showed white and yellow softening, and above the posterior limb of the fissure of Sylvius was an area of central softening measuring 3 cm. in diameter.

DEGENERATION OF MUSCLE FOLLOWING NERVE INJURY.

By FFRANGCON ROBERTS, M.A., M.D.
Captain, R.A.M.C.(T.)
Fellow of Clare College, Cambridge.

CONTENTS.

	PAGE
Chapter I.—Historical Sketch	298
Chapter II.—Polarity of Normal and Degenerated Muscle	303
Chapter III.—Further Observations on Degenerated Muscle	312
Chapter IV.—Condenser Discharges	314
Chapter V.—The Nature of the Degenerative Change	319
Chapter VI.—Electrical Reactions in Conditions affecting Muscles Primarily	338
Chapter VII.—Wasting	341
Chapter VIII.—Conclusion	345

Introduction.

As the result of the large number of cases of nerve injury caused by the present war there has been a remarkable revival of interest in the study of the electrical reactions of denervated muscle. Two years ago, in this country at any rate, few physicians had any accurate knowledge of the subject and this for two reasons, first because injury to peripheral nerves is comparatively rare in civil life and second because in those diseases which are associated with degenerative changes in the muscles routine electrical examination was in the great majority of cases neglected. It is not too much to say that not one student in a hundred had at the time of passing his final examination anything more than the crudest notion of the differences between the electrical responses of a healthy muscle and those of a muscle paralysed by anterior poliomyelitis. At the present day, however, with the presence of thousands of wounded men in the country, this hitherto obscure branch of medicine has leapt into prominence. At no time has a knowledge—and by that I mean a widely-disseminated knowledge—of the elements of electrotherapeutics been so important. To the modern military hospital the electrical department has become as essential a part as the X-ray room.

Many medical men have had to learn the principles of the subject from the beginning, while others who had had previous experience have discovered numerous fallacies in the older teaching. My object in the present paper is to review the present state of our knowledge of the subjects, to put on record fresh observations, and to explain the phenomena as far as possible on rational grounds.

Chapter I.—Historical Sketch: Erb's School.

If we study the history of medicine we find that in cases where one man has possessed knowledge far exceeding that of any of his contemporaries, or has written far more on the subject than anyone else of his time, the tendency is for future generations to accept the teaching of the master as tradition, to ignore the conditions in which he worked, and to neglect to subject his work to the scientific criticism and experiment made possible in later years. A classical instance of this is seen in the way in which the deification of Galen retarded the development of anatomical knowledge for several centuries until the time of Vesalius. Similarly with the branch of medicine now before us—electromyology as it may be called. The dawn of our knowledge of the subject is associated pre-eminently with the name of Erb, who discovered and collected a mass of information bearing upon the change which a muscle undergoes when its motor nerve is divided. And we find that later neurologists have come to regard the name of Erb almost with a reverence like that with which the dissectors of the middle ages regarded the name of Galen.

Now whether we agree or not with the facts laid down by Erb, we must acknowledge the thoroughness with which he and his school investigated the problem. In spite of the great development of electrophysiology in the last forty years our clinical knowledge has advanced comparatively little. Instead, the clinical phenomena have come to be regarded merely as signs, useful for diagnostic purposes, but of an empirical nature, while few attempts have been made to explain them on rational grounds.

The first hint of changes in the electrical responses of muscles after injury to their nerves came from Hallé, of Paris, as early as the end of the eighteenth century. The significance of this discovery, however, was not realized: the time was not ripe and the question lay dormant again for about sixty years. In the middle of the last century came the rapid

growth of the continental school of physiology. It was a time when the electrical phenomena of living matter aroused the greatest interest among biologists. It is not surprising therefore that physicians began to turn their minds in the same direction. To Remak [25] belongs the credit of being the first to revive the forgotten work of Hallé; Neumann showed that a degenerated muscle had lost the power of contracting to a stimulus of momentary duration, while Baierlacher [2] appears to have been the first to give a practical demonstration of clinical cases. In 1859 the last-named showed a case of Bell's paralysis where the muscles gave no response to faradization but were hyperexcitable to the constant current. In 1868 two papers appeared simultaneously, the first by Erb [8], embodying the results of his clinical investigations on man and his experimental work on the rabbit, and second by Ziemssen and Weiss [29], who came to the same conclusions experimentally as Erb. In the following year Brenner confirmed Erb's clinical findings.

In 1874 [9] Erb wrote his classical account of the phenomena. As this account formed the basis of all future work we may give a brief summary of it.

In the first place Erb insisted upon the difference between the changes undergone by nerve and those undergone by muscle. Changes in the former are thus described. Two or three days after section the irritability to the faradic current may be increased; after this the excitability to both faradic and galvanic current diminishes simultaneously, and in seven to twelve days the nerve is totally inexcitable. The change begins at the site of the lesion and spreads peripherally. Return of irritability resembles loss in that it affects both forms of current simultaneously, the process beginning at the lesion and spreading peripherally. The excitability of the nerve remains for a long period subnormal even when there is full voluntary power. In the early stages of recovery there is a period when voluntary power is present, while at the same time the nerve is inexcitable below the lesion but excitable above it—this corresponds to a period histologically when the myelin sheath is either wanting or is incompletely formed.

The changes which muscle undergoes are quite different. Response to faradism diminishes at about the same rate as in nerve, being lost during the second week. This however is true only for stimulation through the skin. Direct stimulation of the bare muscle gives a limited contraction of the subjacent fibres for a long time after the injury. Recovery of faradic irritability usually occurs later than in nerve, remaining subnormal for a considerable period afterwards.

To the constant current muscle becomes less excitable in the first week. In the second week there is a distinct hyperexcitability accompanied by a qualitative change. The contraction which was previously short and brisk is now long and sluggish. Moreover, the muscle becomes increasingly sensitive to the anodal closure current (A.C.C.), so that the anodal closure becomes as effective as the kathodal closure (A.C.C. = K.C.C.) and occasionally ("nicht selten") the A.C.C. preponderates. A similar alteration affects the opening contraction. The muscle may even remain in tonic contraction during the passage of the constant current. When these changes have reached a certain stage, the opening contraction diminishes and, according to Brenner, in the same proportion as the increase of sluggishness and the decrease of irritability to currents of short duration. This stage lasts from three to eight weeks, often longer.

Following this is a second stage characterized by a progressive diminution of excitability to the constant current, a further preponderance of the A.C.C. over the K.C.C., and an increase in the sluggishness of contraction. This is contemporaneous with a gradual disappearance of the cross-striation. Finally a weak A.C.C. is the only "sign of life."

But if regeneration takes place the normal reactions gradually return, but the period during which the excitability is still subnormal may in bad cases be very prolonged.

The return of excitability in muscle is quite independent both of the return of voluntary power and of the regenerative process in the nerve.

A stage can be recognized at which the nerve is undergoing regeneration while the reactions of the muscle are still unaltered—moreover, the clinical picture varies with the rate at which the recuperative process occurs. If regeneration occurs early, normal excitability of the nerves may be co-existent with an abnormal response in the muscle. If the process is later in appearing we have the syndrome of diminished excitability of the nerve with the presence of qualitative changes in the muscle.

Erb also mentions the increased mechanical irritability of the muscle which he says occurs *pari passu* with the heightened excitability to the galvanic current.

A full account is also given of the corresponding histological changes. It is sufficient here to note that he believed in the view then universally held that regeneration of nerve originated peripherally.

The increased irritability of muscle to the constant current corresponds to the period of proliferation of the nuclei of the muscle cells, while the later stage corresponds to the atrophy of the muscle fibres. The slow recovery of the muscle as compared with that of the nerve he believes to be due to the mechanical obstruction afforded by the development of fibrous tissue.

It would not be necessary thus to quote in extenso Erb's teaching were it not for the fact that with the course of years the exact knowledge which existed forty years ago has gradually been forgotten, and all that has percolated into the average text-book of neurology of the twentieth century is the bald statement relating to loss of faradic excitability and polar reversal, the latter expressed in the dogmatic form A.C.C. > K.C.C. Further, many facts which we have quoted above have recently, through ignorance of the early work, been resuscitated and served up as modern discoveries.

On the whole Erb's account met with general acceptance. But there were not wanting those who disagreed on certain points. Of the opponents, most prominent was Vulpian, who dissented from Erb on two grounds, first on the question of the hyperexcitability of muscle to the constant current, second to the predominance of the A.C.C. over the K.C.C. It is in answer to these objections that Erb made his often quoted and often misquoted statement that " he knew scarcely any sign so certain and free from error as the heightened irritability to the constant current and the qualitative changes in the contraction."

The unquestioning acceptance with which Erb's thesis was received in this country is shown by a perusal of Hughes Bennett's work on Medical Electricity [3]. The account which is there given of the signs of degeneration is practically a verbatim report of Erb's account which is summarized above.

On the physiological side Erb's account of the reversal of polarity was received with profound suspicion. As early as 1867 Aeby [1] had shown reversal of polarity in the fatigued frog's muscle. Engelmann, though he at first criticized the experiment, was afterwards forced to admit that the phenomenon could occur. Biedermann, however, was entirely sceptical, believing the results to be due to the secondary anodal and kathodal points inevitable when strong currents are used. Writing in 1894 he says: "Before giving a final judgment it would be necessary here . . . to make further investigations with unassailable methods, for the conditions under which alone experiment can be tried in man, or have been tried in other animals, by no means correspond to the

demands of an exact physiological method. On the other side there are so many results, derived from irreproachable experiments upon different muscles and nerves which are opposed to a theory of reversal of polar effects that any supposed exception must *a priori* encounter suspicion, and can only hope for recognition of the conditions of experiment, and all accessories are perfectly simple and obvious" [4].

In attempting to criticize Erb's work in the light of modern knowledge there are certain points which must be borne in mind. That was a time when electrical apparatus was not to be compared in point of accuracy with that which is available at the present day. It is unfortunate, too, that in his original paper Erb gives no accurate account of the method which he employed. It is clear, however, that he measured the current by the number of Daniell cells in series from which the current is derived—a method open to obvious fallacies. His method of estimating the amount of contraction is indefinite; a slight contraction appears to have been represented by the sign Z, a slightly larger contraction by ZZ, and so on up to ZZZZZ. Ziemssen and Weiss, who give some account of the apparatus which they used, seem to have worked, generally speaking, on the same principles. It must be remembered, too, that these discoveries were made before surgery had been revolutionized by Lister. Operations on nerves must have been of rare occurrence, so that in the great majority of the clinical cases under examination it was impossible to determine the exact form and degree of injury which the nerves had suffered.

Again, it would seem that a large number of the cases studied were those of paralysis of the seventh nerve of the so-called "rheumatic" type. Under ordinary conditions of life this is of all peripheral nerves the one most often affected. Moreover, it is one which on account of its position, and the position of the muscles which it supplies, is most easily studied. Next in frequency to the facial muscles come the intrinsic muscles of the hand. Of the four cases which Erb describes in his original communication, three are cases of Bell's palsy, while the fourth is an injury to the ulnar nerve. As we shall see later, both the facial muscles and the small muscles of the hand and foot are peculiar in their behaviour.

Chapter II.—Polarity of Normal and Degenerated Muscle.

(1) *Unipolar Stimulation.*

The infallibility of polar reversal as an absolute sign of degeneration has recently been challenged by many observers. At the present day, in fact, some neurologists discredit it altogether. With a view to determining whether polar reversal occurs, and, if so, what is the true significance of the phenomenon, I have subjected some hundreds of cases of nerve injury to the test. The first method I employed is that which is commonly followed. One electrode (called the testing electrode) is placed on the muscle near its proximal end. The other (the so-called indifferent electrode) on some distant part proximal to the muscle. Make and break are effected automatically by means of a metronome, thus obviating any source of error due to movement of the testing electrode. The kathode having been placed upon the muscle the current was increased by a rheostat until a minimal contraction appeared. The electrode was then moved about until the most excitable part of the muscle was discovered. The amount of current in milliampères which caused a minimal contraction at this point was read off by an assistant. The current was then reversed, and the minimal exciting current at the same point determined. The corresponding muscle of the opposite side was then tested in the same way. The results are given in Tables I, II and III.

If we first consider Tables I and II, which are taken from cases of injury of the musculospiral and sciatic nerves, we find that excluding Case I, which was examined only nineteen days after injury, with K.C.C. a stronger current was required to evoke contraction on the degenerated side than on the normal side. In the case of A.C.C. the relative excitability on the two sides is variable. Further on the normal side K.C.C. is invariably more effective than A.C.C., while on the degenerated side we have K.C.C. = A.C.C., K.C.C. > A.C.C. or A.C.C. > K.C.C. In the fourth and eighth columns I express the difference as a fraction of the total current. It will be noticed that in the great majority of cases the difference between K.C.C. and A.C.C. is, relatively to the strength of current used, much greater on the normal than on the degenerated side. The small differences are within the bounds of experimental error in the case of paralysed muscles, so that one may say that as a general rule degenerated muscle is indifferent to the polarity of the electrode. Lastly, there is no relationship between the

TABLE I.—Musculospiral Paralysis.

Case No.	Regimental No.	Period after injury	Condition of nerve	Muscle	Paralysed side				Normal side			
					K.C.C.	A.C.C.	Diff.	Diff. K.C.C.	K.C.C.	A.C.C.	Diff.	Diff. K.C.C.
1	2696	19 days	Completely divided ..	Ext. com. digit; ext. oss. met. poll.	5·0 5·0	7·0 5·0	+ 2·0 ..	0·4 ..	7·0 2·5	9·0 4·0	+ 2·0 + 1·5	0·29 0·6
2	10329	39 ,,	,, ,, ..	Ext. com. digit	3·7	3·6	− 0·1	0·03	3·5	4·3	+ 0·8	0·23
3	—	3 months	,, ,, ..	Ext. com. digit; ext. oss. met. poll.	5·0 4·0	5·0 4·0	..	..	3·0 2·0	5·0 3 0	+ 2·0 + 1·0	0·66 0·5
4	2565	3 ,,	,, ,, ..	Ext. com. digit; ext. oss. met. poll.	11·0 10·0	11·0 12 0	..	..	5 0 3·0	6·5 6 0	+ 1·5 + 3·0	0·3 1·0
5	13031	4 ..	Inflamed and adherent	Ext. com. digit	5·0	6·0	+ 1·0	0·2	..	..	..	..
6	15868	52 days	Involved in scar tissue	Ext. com. digit; ext. carp. rad. long.	13·5 12·0	16·0 12·5	+ 2·5 .. 0·5	0·19 0·04	5·1 2·5	8·5 3·5	+ 3·4 + 1·0	0·66 0·4
7	1726	45 ,,	,, ,, ,,	Ext. indicis.. ..	4·5	4·5	..	..	3·0	5·0	+ 2 0	0 66
8	4361	38 ,,	Severely damaged by scar tissue	Ext. com. digit ..	7 0	7·0	..	..	..	..	..	..
9	8044	36 ,,	Involved in scar tissue	,, ,, ..	6·0	6·0	..	..	4·0	5·0	+ 1·0	0·25
10	16289	36 ,,	Scarred: power returning after operation	Ext. indicis.. ..	7·0	7·0	..	..	2·0	4·0	+ 2·0	0·1
11	13036	40 ,,	Not examined surgically	Ext. com. digit; ext. oss. met. poll.	5·0 5·0	4·0 6·0	− 1·0 + 1·0	0·2 0·2	2·6 2·5	3·1 3 5	+ 0·5 + 1·0	0·19 0·4
12	21998	2 months	,, ,, ,,	Ext. com. digit. supin. longus	1·5 2·2	2·2 2·4	+ 0·7 + 0·2	0·47 0·1	1·2 1·0 1·5	1·5 1·6 2·0	+ 0·3 + 0·6 + 0·5	0·25 0·6 0·33
13	1606	3 ,,	,, ,, ,,	Ext. com. digit; ext. oss. met. poll.	10·0 9·8	12·5 10·2	+ 2·5 + 0·4	0·25 0 04	7·0 8 0	8·0 11·0	+ 1·0 + 3·0	0·14 0·37
14	11512	7 weeks	,, ,, ,,	Ext. oss. met. poll; ext. com. digit. supin. longus	9·0 10·0 11·0	9·0 9·0 11·0	.. − 1·0 ..	.. 0·1 ..	2·0 1·5 0·3	3·8 3·3 2 3	+ 1·8 + 1·8 + 2·0	0·9 1·2 6·6

TABLE II.—Paralysis of Great Sciatic Nerve.

Case No.	Regimental No.	Period after injury	Condition of nerve	Muscle	Paralysed side				Normal side			
					K.C.C.	A.C.C.	Diff.	Diff. K.C.C.	K.C.C.	A.C.C.	Diff.	Diff. K.C.C.
15	3023	59 days ..	Complete division ..	Tibial. antic. ..	9·5	10·5	+ 0·5	0 05	5·0	11·0	+ 0·60	1·20
				Gastrocn. ..	13·0	17·0	+ 4·0	0·31	7·0	17·0	+ 10·0	1·40
				Peron. long. ..	21·0	18·0	− 3·0	0·14				
16	—	8 months	Involved in scar; very slight return of voluntary power	Tibial. ant. ..	12·0	12·5	+ 0·5	0·04	8·0	17·0	+ 9 0	1·125
17	851	24 days ..	Not examined surgically	,, ,, ..	8 0	10 0	+ 2·0	0·25	3·0	7·0	+ 4·0	1·33
18	44954	14 weeks ..	Complete division ..	,, ,, ..	20·0	24·0	+ 4·0	0 20	3·0	9·0	+ 6·0	2·0
19	1338	11 months	Involved in scar tissue	,, ,, ..	5·0	5·5	+ 0·5	0·10	2·0	5·0	+ 3·0	1 50
					4·5	4·5	0	0	8·0	12·0	+ 4·0	0·50

TABLE III.—Paralysis of Median and Ulnar Nerves.

Case No.	Condition of nerve	Muscle	Paralysed side				Normal side			
			K.C.C.	A.C.C.	Diff.	Diff. K.C.C.	K.C.C.	A.C.C.	Diff.	Diff. K.C.C
21	Median and ulnar severely damaged	Abduct. poll. ..	1·8	1 9	+ 0·1	0·05	0 8	0·8	..	..
	Resection and suture ..	Dorsal interossei ..	3·7	5·1	+ 1·4	0·38	2·4	2·2	− 0·2	0·08
22	Median: involved in scar tissue	Abduct. poll. ..	2·3	2·3	..	..	..	..	..	..
23	Median: complete division	,, ,, ..	4·5	4·1	− 0 4	0 09	3·0	3·3	+ 0·8	0 27
							3·0	4·1	+ 1·3	0·43
24	Median: involved in scar tissue	Oppon. poll. ..	9·5	5·0	− 4·0	0·42	8·0	9·0	+ 1·0	0·125
25	Ulnar: involved in scar tissue	Dorsal inteross. ..	8·0	10·0	+ 2·0	0·25	6·0	10·0	+ 4·0	0·66
26	Ulnar: no operation ..	,, ,, ..	6·0	8·0	+ 2·0	0·33	7·0	6·0	− 1·0	0·14
27	,, ,, ,,	,, ,, ..	10·0	6·5	+ 3·5	0·35	5·0	5·0	..	..

Notes to Tables I, II, and III.

(1) The figures indicate the current in milliampères required to produce minimum contraction.

(2) In the fourth and eighth columns of figures I have represented the difference as a fraction of the total current (K.C.C.).

(3) In the third and sixth columns of figures a + sign indicates K.C.C. > A.C.C.: a − sign indicates A.C.C. > K.C.C.

(4) In the cases where the nerve was not examined surgically, evidence of severe injury was indicated by the absence of response to Faradism and the sluggish response to the constant current.

polarity of the muscle and the degree of injury which the nerve has sustained.

Table III shows the results obtained from cases of injury to the median and ulnar nerves. It will be observed that all the muscles examined are the intrinsic muscles of the hand. Again we see the variable K.C.C. : A.C.C. relationship obtaining in the degenerated condition, but in these cases we find the same variability on the normal side, reversal of polarity occurring with approximately equal frequency on the two sides.

(2.)

In stimulating the motor point of a normal muscle we are dealing with a nerve-muscle complex, the current affecting three different "receptive substances." There are the intramuscular branches of the nerve, and there is the muscle tissue proper. In addition, Langley [15] has shown that there exists in between yet a third distinct substance, existing as a kind of synapse between nerve and muscle. This has been confirmed by Keith Lucas [18], who finds that these three receptive substances can be distinguished by their excitability. The least excitable (Lucas's α substance) being the muscle proper, and therefore present equally in all parts of the muscle, the most highly excitable (β) being the substance at the synapse or neuro-muscular junction, while the purely nervous substance (γ) is intermediate in excitability. With one electrode placed over the motor point of the muscle the three substances will be affected to a varying extent depending upon their excitability. The β substance will therefore preponderate, and we must regard the mechanical effect as the result mainly of stimulation of this substance.

Now, there is one point which must be mentioned here, since it is too often forgotten in clinical work, and that is the difference between the real and the apparent electrodes. Supposing we place the negative electrode on the motor point of the biceps and the positive electrode on the side of the neck. On closing the circuit the current flows into the body at the neck and leaves it at the arm. But it would be absurd to say that the kathode is at the muscle and the anode on the neck. The true anode is the point or points at which the current enters the *muscle*, and the true kathode is the point or points at which it leaves the *muscle*. As the current leaves the positive electrode at the neck it spreads out diffusely according to the resistance offered by the tissues, it enters the muscle diffusely, and in the case before us where the

muscle is large and superficially placed, it leaves the muscle concentrated at the point nearest to the negative electrode. This is shown diagrammatically in figs. 1 and 2. The negative field is the more concentrated, and will exert a greater physiological effect than the more diffuse positive upon the neuro-muscular substance, since this is in the immediate neighbourhood of the former. On reversing the current we get the opposite state of affairs. The current enters the muscle (anode)

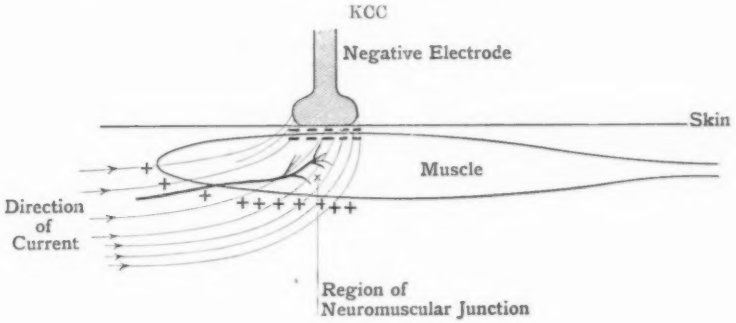


FIG. 1.

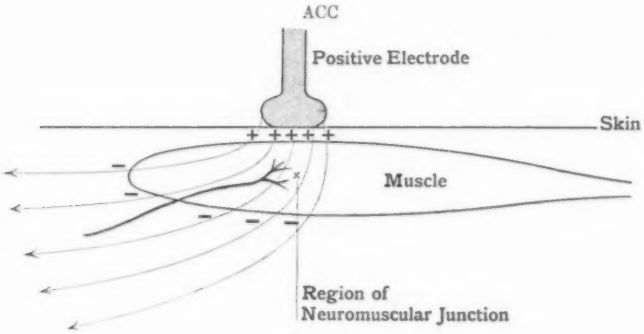


FIG. 2.

concentrated in the region of substance β, leaves it diffusely (kathode), the anode therefore exerting the greater effect. Now if we test the polarity of a normal muscle at different points on its surface we find that the normal condition K.C.C. > A.C.C. is obtained with constancy only at the motor point. At other parts of the muscle the relationship is variable. This is shown in Table IV.

TABLE IV.—Effect of Varying Position of Electrode.

Case (28) *of Complete Division of Sciatic Nerve. Muscle—Tibialis anticus. The figures indicate amount of current required to produce minimal contraction.*

Position of testing electrode	Paralysed limb		Normal limb	
	K.C.C.	A.C.C.	K.C.C.	A.C.C.
Proximal end of muscle	15	15	1·5	5·0
Middle of muscle	15	15	4·0	6·0
Distal end of muscle	11	14	10·0	9·0

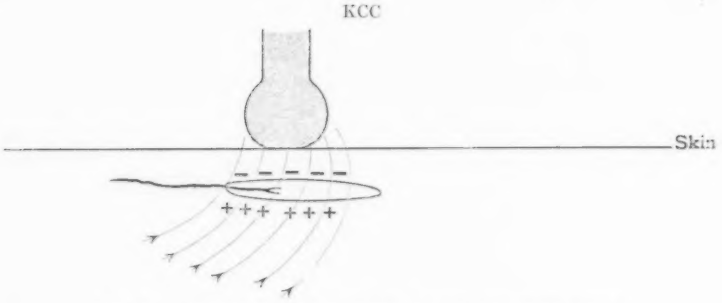


Fig. 3.—True anode and kathode in the case of a short muscle.

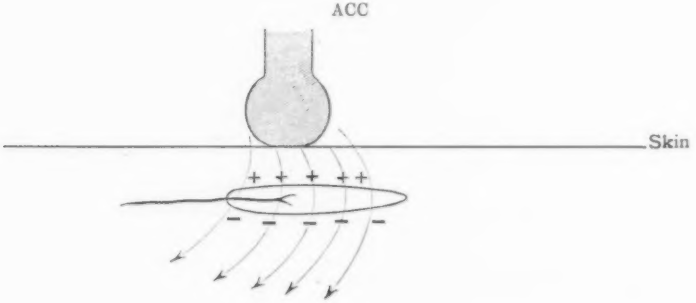


Fig. 4.

When degeneration occurs the *first* change is the disappearance, as we shall see later, of the physiological "motor point"—all parts of the muscle becoming equally excitable. From these facts one may conclude that the indifference to polarity which we have seen is characteristic of the degenerated condition, is due to the absence of the

neural and neuro-muscular substances, the muscle proper responding directly to the stimulus.

It now remains to explain the anomalous behaviour of the normal muscles in Table III. All the muscles here are of very small size compared with those of Tables I and II. In testing these small muscles it is a matter of much greater difficulty to single out the motor point and to obtain a concentrated effect at the point of entry or of exit. The current enters the muscle diffusely and leaves it diffusely. Consequently we cannot tell whether the muscle is being stimulated at the point of entry or at the point of exit of the current. This is shown in figures 3 and 4.

It is sometimes stated that in testing the intrinsic muscles of the hand the so-called indifferent electrodes should be placed on the opposite side of the hand in testing the dorsal interossei, for instance, the indifferent electrode should rest against the palm of the hand. This practice I believe only aggravates the anomalous behaviour of these muscles, since the current is thus being made to pass at right angles to the direction of the muscle fibres. I have obtained more consistent results by placing the indifferent electrode on the arm.

(3) *The Longitudinal Reaction.*

It has long been known that in a normal muscle contraction is most easily evoked at the motor point while in degenerated muscle, it can often be activated more easily at the distal end. This was called the Longitudinal Reaction. This, as Lewis Jones [12] pointed out, was due to the destruction of the neural elements, leaving the muscle equally excitable at all points of the surface. This effect is, however, obtained only when the indifferent electrode is placed on some part of the body proximal to the muscle, and this for a very simple reason. When the testing electrode is placed distally the muscle is brought under the influence of the current as shown in figures 5 and 6. Consequently as all parts of the muscle are equally excitable the physiological effect will be greater. But in the normal muscle the motor points being much more irritable than any other part of the muscle, this effect is not seen.

(4) *Effect of Increasing the Stimulus upon Polarity.*

If instead of taking minimal contractions as our guide, we study the changes in the K.C.C. : A.C.C. relationship a further remarkable

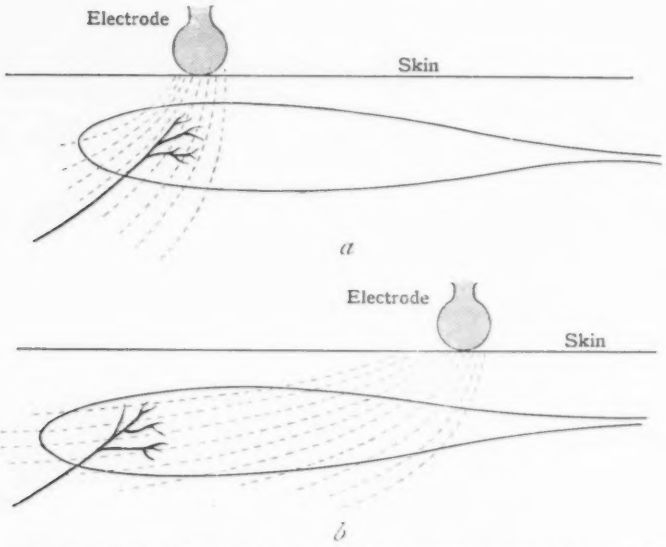


FIG. 5.—Illustrating the longitudinal reaction. In the normal muscle *a* is more effective than *b*, owing to the condensation of field at the motor-point in the former.

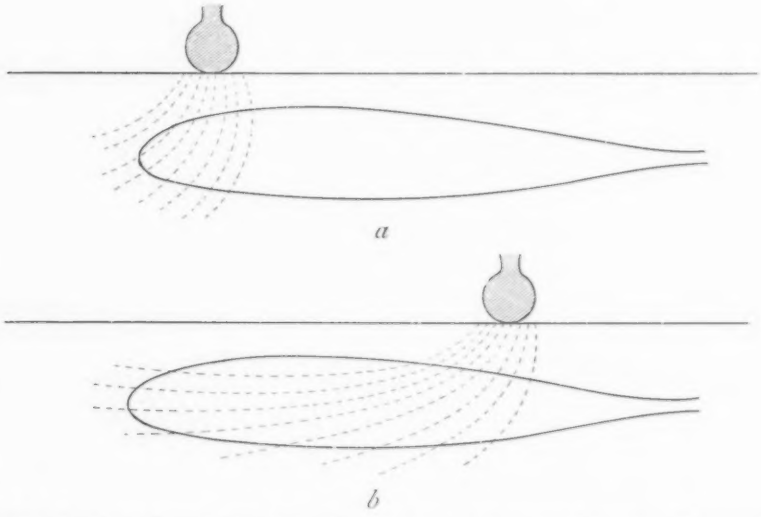


FIG. 6.—Illustrating the longitudinal reaction in the degenerated muscle. The muscle being equally excitable all over, owing to the absence of the "motor-point," *b* is more effective than *a*, since in the former more of the current traverses the muscle.

difference in the behaviour of normal and degenerated muscle is seen. Taking the healthy muscle first we find the amount of current required to give minimal contractions with K.C.C. and A.C.C. With the kathode on the muscle we then increase the current until a full contraction is obtained. This having been recorded, the current is then reversed, and the current required to evoke an equal contraction is determined. The degenerated muscle is then tested in the same way.

TABLE V.—Effect of Increase of Stimulus on K.C.C. : A.C.C. Relationship. Muscle—Tibialis anticus.

Case No.	Case	Limb	Minimal contraction			Full contraction		
			K.C.C.	A.C.C.	Diff.	K.C.C.	A.C.C.	Diff.
29	Pte. McC. ..	Normal limb	5	11	6	14	25+	9+
		Degenerated limb ..	Stimulation painful					
30	Pte. W. ..	Normal limb	2	5	3	7	13	6
		Degenerated limb ..	15	20	5	35	37	2
31	2nd Lieut. S. ..	Normal limb	5	8	3	10	17	7
		Degenerated limb ..	17	17	0	—	—	—
32	Pte. McD. ..	Normal limb	5	11	6	13	22	9
		Degenerated limb ..	10	12	2	20	20	0
33	—	Normal limb	3	9	6	4	12	8
		Degenerated limb ..	20	24	4	—	—	—

Table V gives the result of the examination of five cases in this way. It will be noticed that in the case of the normal muscle, as the strength of contraction increases, the difference between K.C.C. and A.C.C. also increases. In the degenerated state, on the other hand, increasing the force of the contraction does not materially affect the polarity—the difference being small in any case.

(5) *Bipolar Stimulation.*

A muscle can also be stimulated by placing both electrodes over it, one at either end. I have tested this method and find that the results obtained are almost exactly the same as are obtained by the unipolar method. With the kathode at the proximal end and the anode at the distal end the current is descending the muscle and vice versa. Owing to the propinquity of the motor point to the proximal end we have in normal muscle—

Descending > Ascending.

In the case of degenerated muscle, the relationship is variable, but any difference is comparatively slight.

Summary of Chapter II.

(1) In the case of long muscles in the normal condition we find K.C.C. > A.C.C. This is constant.

(2) In degeneration the muscle is almost indifferent to polarity. This is due to the disintegration of the neural and neuro-muscular elements at the motor point.

(3) The normal K.C.C. > A.C.C. is only obtained with constancy when the testing electrode is placed over the motor point.

(4) In normal short muscles such as those of the hand or face, the difficulty of singling out the motor point is responsible for the inconstancy of the results obtained.

(5) The "longitudinal reaction" as usually tested is due to the greater amount of current traversing the muscle when the testing electrode is distal than when it is proximal.

(6) In normal muscle the K.C.C. : A.C.C. difference increases with increase of stimulus. In degenerated muscle the response is indifferent whatever the strength of current.

(7) The "bipolar" has no advantage over the "unipolar" method.

Chapter III.—Further Observations on Degenerated Muscle.

(1) *Increased Galvanic Response.*

We have seen that the quantitive change in the response to the constant current was the subject of dispute between Erb and Vulpian. Erb asserted the invariable increase in galvanic excitability, beginning in the second week and lasting as long sometimes as two months. But he afterwards modified this statement, admitting that in some cases of paralysis of the lower limbs the phenomenon did not appear. Experimenting upon rabbits in which the bare muscle is stimulated, I have found this to be a constant sign; about three weeks after nerve section, the increased response to the galvanic current with the diminished response to the faradic current is indeed very striking. In clinical work on the other hand the results obtained are less constant. A stage of increased excitability to the constant current can sometimes be recognised, but not always. The fact is I believe theoretically such a stage always occurs. I have already called attention to the imperfections of apparatus used in Erb's time. Even with all the improvements of the present day we still have no means of estimating the strength of current which is actually stimulating the muscle as

distinguished from the strength of current which is passed into the body.

Owing to the impossibility of making stimuli which are physiologically equal on the two sides of the body, no true quantitative estimate of excitability can be made. I regard then the increased galvanic response as a constant and necessary part of the reaction of degeneration in an early stage, but one which cannot always be demonstrated owing to imperfections of technique. At the same time it is possible that the phenomenon is not so well developed in human muscles as in those of the rabbit.

(2) *Absence of Response to the Constant Current.*

Degenerated muscle may respond to the constant current for a very long time. Sherren [27] quotes a case of complete division of the musculo-spiral nerve in which the muscles responded twenty-three years after the injury. It would appear that a response is obtained as long as any contractile material remains, that is to say until the muscle has completely atrophied, or has been replaced by fibrous tissue. As however there are certain conditions under which the constant current fails to evoke any contraction, these must be described.

The average adult patient can usually tolerate the galvanic current of strength sufficient to cause contraction of the muscle. In some cases of course owing to temperamental conditions or, in cases of incomplete injury to referred pain, caused by the irritation of the nerve trunk it becomes difficult to bring the required strength of current into play. There are too, certain regions of skin where the electric current is particularly unpleasant. I have found the front of the thigh to be of this nature, making the examination of the quadriceps extensor and sartorius often very difficult.

The presence of œdema has a very marked effect upon the response. A small excess of fluid in the subcutaneous tissue at the point of application of the electrode is sufficient to prevent the contraction even of normal muscle to either form of current. The reason for this no doubt is that the subcutaneous fluid is a better conductor than the muscle. The current flows along the line of least resistance without touching the deeper structures.

Failure to respond to the constant current also occurs when the muscle itself is damaged. This is a point of some diagnostic importance. Recently a patient was brought to me suffering from a gunshot wound

of the forearm. All the muscles responded normally except the flexor longus pollicis which gave no reaction whatever. In this case the muscle itself was damaged, since had the typical "reaction of degeneration" been obtained it would have signified injury to the branch which the median nerve supplies to this muscle.

(3) *Return of Faradic Excitability.*

As we have seen in our historical survey, it was taught in the seventies that voluntary power returns before the response to the faradic current. Some recent observers, however, have found that this is usually but not always the case. In my own experience voluntary power returns first in normal cases. Usually the muscle attains to considerable strength before it responds to the interrupted current. Cases in which response to faradism occurs while still completely paralysed come into two categories. There are those where the patient has never been encouraged to exert his will power and has never even tried to use the muscles. In other cases there is a "functional" element superadded to the organic mischief. These cases never fail to respond to encouragement and other appropriate measures.

Summary of Chapter III.

(1) Increased galvanic excitability is a necessary stage in the process of degeneration. It cannot however always be recognized. It may be less evident in man than in the rabbit.

(2) Absence of galvanic response occurs: (1) in cases of very long standing; (2) as a result of hypersensitiveness of the skin; (3) when œdema is present; (4) when the muscle itself is injured.

(3) In regeneration, voluntary power returns before faradic excitability, except in functional cases and those in which the extent of voluntary power is not realized by the patient.

Chapter IV.—Condenser Discharges.

In the early days of electromyology it was known that degenerated muscle failed to respond to the faradic current because the individual impulses were of so short duration. Many attempts have since been made to stimulate muscles by known quantities of electricity, and so to estimate different grades of degeneration according to the minimum duration of current to which the muscle responds. For this

purpose condensers were introduced, first by Boudet in 1888; their use was afterwards extended by Dubois [7]. In this country the chief exponent of this system of testing was Lewis Jones, who introduced a set of twelve condensers, the capacity and discharge duration of which, at a voltage of 100, are these :—

Capacity in microfarads	Duration of discharge
0·016	$\frac{1}{24000}$ sec.
0·025	$\frac{1}{16000}$,,
0·05	$\frac{1}{8000}$,,
0·62	$\frac{1}{6000}$,,
0·08	$\frac{1}{4800}$,,
0·125	$\frac{1}{3200}$,,
0·25	$\frac{1}{1600}$,,
0·33	$\frac{1}{1200}$,,
0·5	$\frac{1}{800}$,,
0·66	$\frac{1}{600}$,,
1·0	$\frac{1}{400}$,,
2·0	$\frac{1}{200}$,,

A further modification has recently been introduced by Hernaman-Johnson [11], by which the voltage can be varied. It is supposed that, by finding the smallest charge which will cause stimulation, an absolute value can be given to the stage to which degeneration has advanced and that day to day tests give a measure of the progress of the case.

Let us now consider whether the condenser gives that exact scientific information which it is alleged to give and whether we can obtain by its means any information of value which cannot be got by means of the faradic and galvanic currents.

Of the constant nature of the capacity of a given condenser there can of course be no question. But before we can consider the condenser as an instrument of exact scientific measurement we must have some means of finding what proportion of the charge passes through the muscle and stimulates it. To say that in the ordinary process of stimulation the whole charge is effective is absurd. The muscle responds to that part of the charge which passes through it, not to the whole charge which passes through the body. At present we have no means of estimating the amount of the part which is active. Comparison with the corresponding muscle of the healthy limb, although some sort of guide is open to the same fallacy as in the case of the constant current—that is to say, the varying conditions on the two sides—the size of the muscle, the state of the subcutaneous tissue. Further, if the result obtained at one examination of the muscle cannot be regarded as a definite standard, it follows that the results obtained

from a series of examinations at intervals of some weeks or months will have still less value. Besides the condition of the muscle many other factors will be different on the two occasions—the position of the electrodes, the amount of subcutaneous fat and the temperature of the skin, in spite of (or in consequence of) artificial attempts at keeping the skin warm. With so many varying factors the amount of electrical energy which is available for the stimulation of the muscles must be subject to very considerable variation. So far from being more scientific than the constant current method, it is in reality less so, for by estimating the relative excitability to K.C.C. and A.C.C. we have only one variant—the direction of the current. We are here comparing the effect of a given current with that of the same current reversed, all other factors being unaltered.

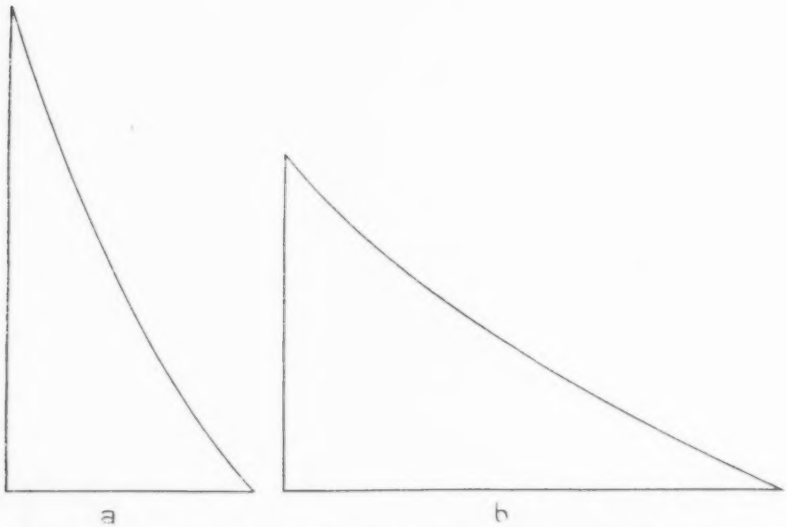


FIG. 7.—Discharge of condenser: *a*, through low resistance; *b*, through high resistance.

Those who advocate the use of the condenser lay great stress on the fact that with increase of the capacity of the condenser, voltage being constant, there occurs a proportional increase in the duration of the discharge. But the discharge duration, so far from being constant, varies with the external resistance of the circuit.

Figs. 7 *a* and *b* show the difference in the form of discharge with small and with high resistance. It is idle to suppose, as has been

supposed by some, that the resistance of the body is a negligible quantity. A short experience of passing current through the body will be sufficient to convince anyone that the resistance offered by the body is considerable, quite sufficient at any rate to alter the duration of discharge. But even if the duration were independent of any external circumstance it is difficult to see what scientific value can be attached to its expression. We have already seen that the stimulating effect of the constant current depends upon two factors, strength (voltage) and duration, which are related inversely. Further, there is a threshold voltage below which contraction does not occur even with infinite duration. Consider the discharge A—B. The current falls rapidly to

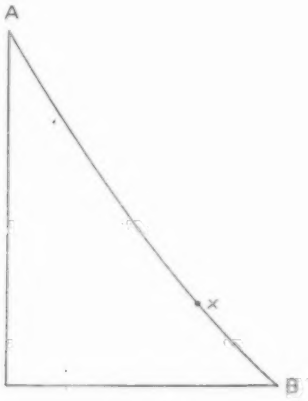


FIG. 8.

zero at B. At a certain point x it reaches the minimum effective voltage. The part xB of the current is therefore totally without physiological effect, and since we have no means of knowing the exact position of the point x we cannot tell the duration of the effective current. Moreover, as the inclination of the curve varies with resistance so the distance of x from A and B will vary, a fact which further complicates the question.

Another factor which must be taken into account is the direction of the current in relation to the direction of the muscle fibres. Leicher [17] showed that with a given current the maximum effect is produced when the current traverses the muscle longitudinally. If the current passes absolutely at right angles to the direction of the fibre it is

without effect. The position of the electrodes, therefore, is all important if accuracy is the objective. In the method adopted by some, namely, pinching up the muscle between the electrodes, the electrical discharge is acting at the greatest physiological disadvantage.

It must be admitted, however, that the condenser discharge possesses one great advantage over the galvanic current in that it is comparatively painless, but as a set-off against this must be stated the undoubted fact that the condenser discharge spreads more diffusely in its passage between the electrodes, and consequently it is more difficult to limit the effect to one muscle.

A more scientific method was introduced in 1903 by Leduc, who invented a means of interrupting the constant current mechanically in such a way that the duration of current as well as the interval between could be altered. Both were capable of measurement. But the apparatus was so complicated that the method never came into general use in this country.

Even if the condenser did give an absolute value it is doubtful if the results would be of any constant practical use as a test of recovery. We have seen that usually voluntary power returns before the faradic response. If the testing is performed by means of the condenser it will be found that voluntary power will usually have begun to return before there is any change in the electrical response. Here then the condenser possesses no advantage over the constant current.

In conclusion, we may say then that the condenser does not give the accurate estimate of degeneration which is attributed to it. It is indeed no royal road to diagnosis and prognosis. It may in some cases be of use in supplementing the information obtained from the older methods, but these on the whole are preferable. The animal body is from an electrical point of view terra incognita, the mysteries of which are not to be explored by the further elaboration of apparatus in the direction of obtaining graded stimuli, the accuracy of which is upset by a varying and unknown resistance.

Summary of Chapter IV.

(1) The defects of the condenser are that the discharges vary with the resistance and with other factors. They do not give uniform results either in theory or in practice. It is difficult to confine the discharge to any individual muscle.

(2) The one advantage of condenser discharge is that they do not cause pain.

Chapter V.—The Nature of the Degenerative Changes.

When we come to consider the changes following section of a motor nerve we are forced to admit that in the past investigation has been directed far more towards the changes occurring in nerve than towards those occurring in muscle. The controversy upon the regeneration of nerve has been partly responsible for this. But the process of degeneration does not stop short at the nerve-ending, the regressive changes in muscle are something more than disuse atrophy following paralysis of the nerve. Clearly, the disintegration of the nerve-endings within the muscle, although it may affect the degree of response of the muscle to artificial stimuli, cannot by itself explain the striking change in the form of the contraction. This must be due to some change in the muscle itself.

The muscle-fibre no less than the nerve-fibre is dependent upon its connection with a healthy anterior cornual cell for the maintenance of its normal structure and excitability. This is not surprising when we remember that in the earliest metazoa contractile tissue first appears in the form of fibrils laid down in a cell another part of which is adapted to the reception of stimuli, the whole cell being dependent functionally upon the nucleus. The same cell is receptor, conductor and effector, to use Sherrington's nomenclature. In higher forms, in spite of the increasing complexity of the reflex arc, the central part of the mechanism exerts upon the effector organ an influence which involves more than the mere discharge of impulses. The anterior cornual cell is still the trophic centre of the muscle-fibre as well as of the nerve-fibre. Judged from this point of view the muscle is merely a special modification of one end of the conductor mechanism as the receptor organ is at the other.

We shall now proceed to consider why, after section of a nerve, the brisk normal twitch gives place to the sluggish response characteristic of degeneration.

(1) *The Process of Degeneration in White and Red Muscles of the Rabbit.*

In striped muscle, fibres of two kinds are found, the white and the red. These are distinguished from one another histologically and physiologically. In structure the red fibres have a greater amount of sarcoplasm than the white, and have also a certain peculiar arrangement of blood-vessels first pointed out by Ranvier [23], the significance

of which is not known. On stimulation white fibres respond briskly while the red fibres respond with a sluggish contraction after a comparatively long latent period. In man each individual muscle is composed of a mixture of both kinds of fibres, while in some animals, notably the rabbit, different muscles are composed exclusively of one kind or the other, the gastrocnemius for instance being purely white, and the soleus purely red.

Now the contraction of normal red muscle bears at first sight a resemblance to that which is obtained from degenerated human muscle. It might be thought, therefore, that the reaction of degeneration as found in man is due to the process of degeneration affecting the white and red fibres at unequal rates, the former degenerating more rapidly than the latter, so that at a certain stage in the process the red fibres alone respond to electrical stimulation. In other words, the reaction of degeneration is brought about by the unmasking of the red fibres owing to the rapid death of the white kind.

But that this hypothesis of degeneration is improbable is shown by the following considerations:—

(1) The resemblance between the mixed degenerated muscle of man and the pure red muscle of the rabbit is more apparent than real. In the degenerated condition response to induction shocks is very difficult to obtain, even though at the same time the excitability to the constant current may be considerably exalted. In the normal soleus of the rabbit the single induction shock is just as efficient a stimulus as the constant current. Moreover, this is still the case after the administration of curare, showing that stimuli of momentary duration affect this type of muscle directly and not through the nerve endings.

(2) This hypothesis presupposes the more rapid degeneration in the white fibres than in the red. It has been stated by Joteyko that such is the case, but after a careful examination of several rabbits I am unable to find any foundation for this statement. If there is any difference in the behaviour of the two varieties of fibre it would appear in the opposite direction. According to my results the red muscles degenerate in the majority of cases more rapidly than the white. If we take loss of weight as an indication of the rate of degeneration the results shown in Table VI (p. 342) indicate that there is no constant difference in the amount of wasting shown by the gastrocnemius and soleus. I admit, however, that this is a poor criterion, since atrophy is no doubt partly due to disuse.

(3) The degenerative process in the pure white muscle corresponds fairly closely to the process as it affects the mixed muscle of the human being. It would appear, therefore, that in the latter it is always the white fibres which respond whatever may be the stage of degeneration. Normally the white fibres being more excitable, and responding more briskly than the red, determine the rate of contraction of the whole muscle. The process of degeneration would, therefore, seem to be due to changes occurring in white fibres mainly.

(4) The "reaction of degeneration" is a change which occurs not slowly as may be supposed from its name, but with comparative suddenness, as has recently been shown by Paton and Findley [22]. It is difficult to explain this by assuming that the white fibres become suddenly inexcitable some few days after section of the nerve.

Although we cannot in this way explain the phenomena of degeneration as they occur in man, the comparative study of the electrical behaviour of the two varieties of muscle fibre does throw some light on the problem before us.

I have followed the process of degeneration in the muscles of the rabbit's hind leg after resection of a portion of the sciatic nerve in the thigh. Rabbits were examined at different periods ranging between four days and four months after neurectomy. In all cases the left nerve was cut, the right side being used as a control. In each case before the muscles were investigated the condition of the nerve was examined to ensure that the operation of neurectomy had been effective. The muscles were stimulated by means of small sponge electrodes, soaked in salt solution, which were laid on the bare muscle. Where it was desired to take tracings the tendon was cut, the muscle freed from the surrounding tissues, except at its proximal attachment, and the distal end was attached to the recording lever.

(2) *Electrical Behaviour of White Muscle.*

The excitability to the interrupted current diminishes rapidly, but does not disappear until many weeks have elapsed. As long as three months after section of the nerve a slight contraction can be obtained, using very strong currents. It is difficult, however, as late as this to produce a contraction of the muscle; as a rule, all that occurs being, as Erb pointed out, a contraction of the fibres adjacent to the electrodes. Experimentally, therefore, faradic response is not an absolute test of degeneration, though clinically it is so owing to the limitations of stimulation due to the resistance of the skin and human powers of

endurance. Erb mentions a case where, in spite of an injury to the nerve, faradic response was present. I have never come across such a case and am very doubtful whether it can occur after complete division, sufficient time of course having elapsed to allow of degeneration.

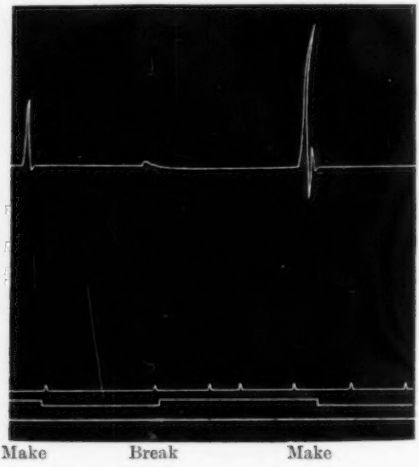


FIG. 9. –Tibialis anticus. Eight days after section of nerve. Tonus has not yet appeared.

The excitability to the constant current is unaltered as far usually as the eighth day (fig. 9), a brisk twitch occurring at make, and if the current be not too weak, at break, the muscle remains uncontracted while the current is passing. During the second week two well-marked changes occur. The first is the increased excitability to this form of current, coincident, it must be remembered, with a diminished response to the faradic current. At the same time the phenomenon of galvanotonus appears—the muscle remains contracted while the current is passing. This was first demonstrated by Fick [10] for degenerated amphibian muscle.

(3) *Characteristics of Galvanotonus.*

Fig. 10 shows the condition found in the gastrocnemius fourteen days after section of the nerve. With a weak stimulus (fig. 10, I) the contraction both at make and at break is of a double nature, the first being a twitch just as brisk as the response of the normal muscle, the second a slower contraction which appears after the first. In the

tracing this appears first as the arrest of the relaxation following the twitch. On increasing the current the slower contraction becomes further developed until it forms a tonic contraction which persists during the passage of the current. At break there is again a twitch before relaxation, the lever rising as it were above the plateau of the persistent contraction.

The question now arises whether the second element in the contraction is due to the rapid change of potential as in the case of the primary twitch or whether it is due to the persistence of the current.

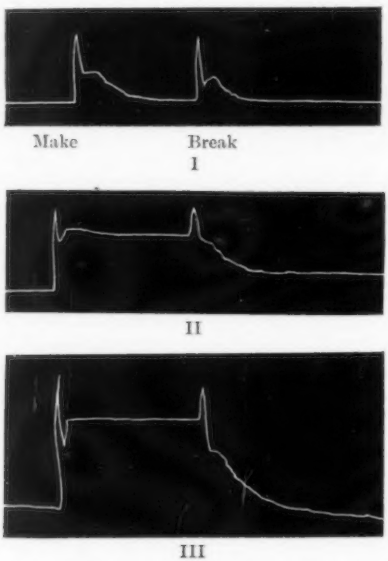


FIG. 10.—Rabbit's gastrocnemius fourteen days after section of sciatic nerve, showing effect of increasing the stimulus.

A glance at fig. 10 I shows that the contraction at break is of a double nature just as the contraction at make. Rapid change of potential is therefore the cause of the second part of the contraction as well as of the first.

If the degenerated muscle is stimulated with a weak single induction shock, the result is a simple twitch (fig. 11 *a*). With stronger shocks the second element appears, but is not well marked (fig. 12). This shows that with currents of momentary duration the main effect is

twitch, the second element being less prominent than when currents of longer duration are used.

We must now inquire whether the mere passage of a constant current is of itself sufficient to evoke contraction in a degenerated muscle. In order to determine this point the gastrocnemius in the

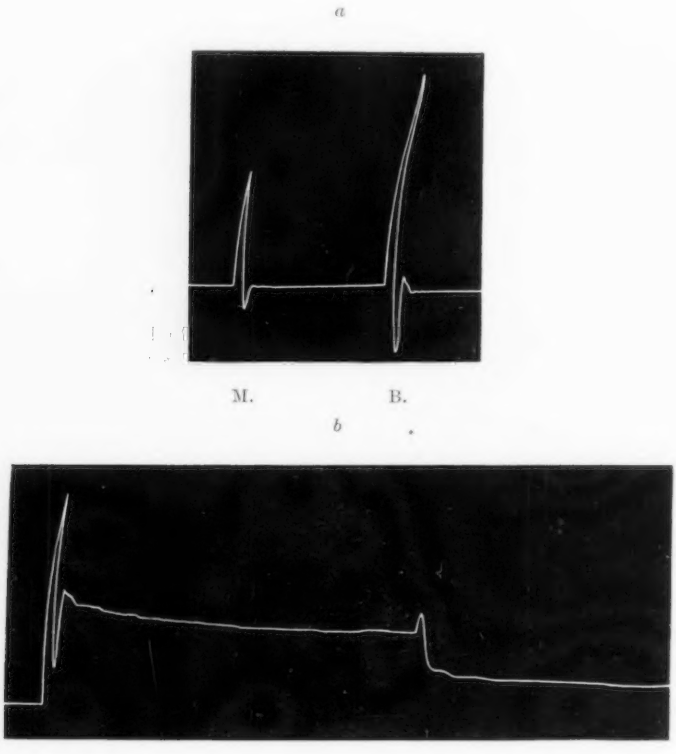
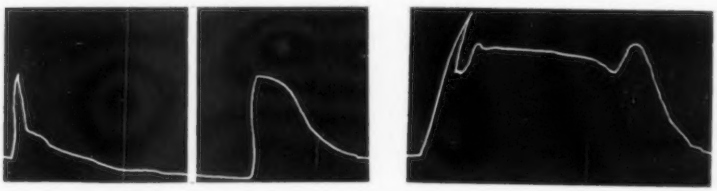


FIG. 11.—Tibialis anticus, twenty-one days after section of nerve. Upper diagram: Two induction shocks. Lower diagram: Constant current. Time: $\frac{1}{2}''$ = 1 second.

fifteenth day of degeneration is stimulated by the make of a weak current insufficient to cause galvanotonus. This causes a contraction (e.g., fig. 13). When the muscle has completely relaxed, the current is increased by lowering a resistance placed in the circuit. The muscle is unaffected. At break of the current (*b*) contraction occurs. The

muscle is then stimulated with a current of the same strength as at *b*. There is a well-marked galvanotonus (*c-d*). The mere passage of a current is therefore insufficient to cause contraction of a degenerated muscle. Change of potential of a certain rapidity is necessary to cause contraction, the flow of the current maintaining a condition which it cannot itself initiate.

FIG. 12.—Degenerated white muscle. Strong induction shock.



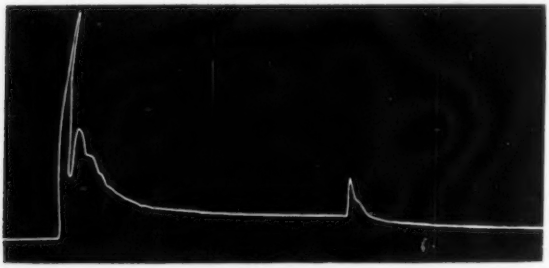
a *b* *c* *d*

FIG. 13.—Gastrocnemius, fifteenth day. *a*, Contraction obtained by make of a weak current; the current passing through the muscle was then gradually increased; this increase had no effect. At *b* the current (now strong) was broken. *c*, Contraction obtained by make of current of same strength as at *b*. Note definite galvanotonus.

(4) *Effect of Fatigue on Galvanotonus.*

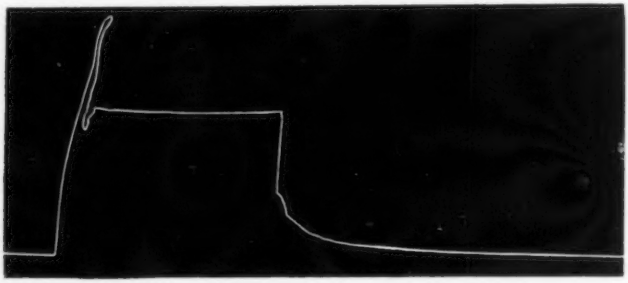
The two components of the process of contraction are capable of further differentiation. Fatigue for instance affects them unequally. If the gastrocnemius preparation be stimulated by several induction shocks in rapid succession we find that on passing in a constant current immediately afterwards the galvanotonus is still as well marked as it

was at first. If the same muscle be stimulated repeatedly by make and break of the constant current, then tonus diminishes and disappears, leaving the initial twitch unaffected. This is shown in fig. 14. Here the tibialis anticus, twenty-one days after section of the nerve, was stimulated with sixty induction shocks—this left tonus unaffected.



M. B.

FIG. 14.—Tibialis anticus twenty-one days after section of nerve. Stimulation by constant current after stimulating with constant current about sixty times. Note fatigue effect.



M. B.

The same muscle stimulated by constant current after stimulating with about sixty induction shocks. Note the absence of fatigue effects. Time: $\frac{1}{2}'' = 1$ second.

It was then stimulated sixty times with the constant current, the duration of the current being about one second each time. The second part of the contraction is less marked and tonus is absent, whereas the twitch at make is unaffected. The second component is therefore easily fatigued by currents of long duration, whereas it is unaffected by currents of momentary duration. By the constant current the slower contraction is more easily fatigued than the twitch.

(5) *Effect of Load.*

If we consider the effect of load upon the form of contraction we find that on the whole the effect of increasing the load is to diminish both parts of the contraction together. This is shown in fig. 15. With each load the muscle made one contraction, fatigue being thus reduced to a minimum. It will be seen that while the height of contraction at make and break is diminished, tonus is still well maintained, though at a lower level.

(6) *Combined Effect of Fatigue and Load.*

After having performed this series of weight-lifting experiments, the same muscle was then given a load of 70 grm. once more. Now the relative heights of the two components of the contraction are subject to great variation, not only at different stages of degeneration and in different muscles, but also in one muscle at the same experiment. On this occasion we see (16 *a*) that the maintained contraction is at a considerably higher level than the summit of the initial twitch, the summit of which is marked by the slight kink at the level B. Beyond this the lever rises more slowly until the plateau is reached. When the current had been broken the constant current was then passed in twelve times the duration, each time being about one second. Immediately

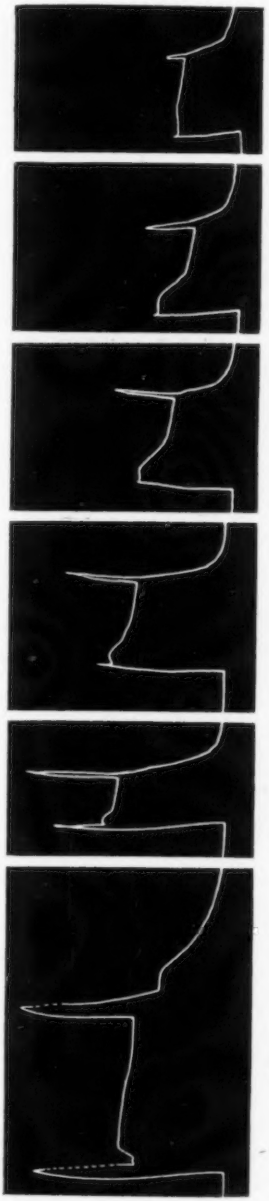


FIG. 15.—Degenerated white muscle. Effect of load upon galvanotonus. In the first figure the drum is moving more rapidly than in the others.

after this another record was taken, shown in 6 *b*. We see that there is just the initial rise (A′—B′), which is not very different from the initial rise in the first case (A—B). Following this however the two curves are very different, for now the slower contraction is not as high as the initial twitch and the tonus is very considerably diminished. We see therefore that although degenerated muscle has the power of maintaining a load in the contracted state during the passage of the constant current, yet when the muscle has performed real mechanical work in lifting the load several times against gravity, this power is considerably impaired, the actual lift at make being unaffected.

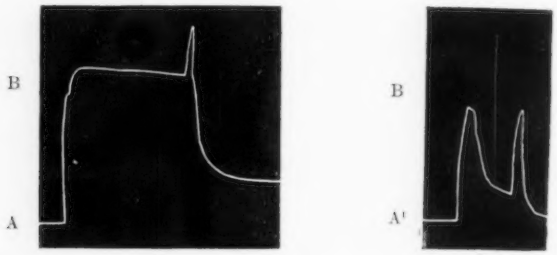


FIG. 16.—Degenerated white muscle. Combined effect of load and fatigue. *a*, Effect of constant current. Load = 70 grm. *b*, The same after passing in the constant current twelve times, the duration of the current in each case being about one second. Note the effect on tonus.

(7) *Electrical Behaviour of Red Muscle.*

In the normal red muscle a single contraction is, as we have said, more sluggish than that of the white muscle, and is preceded by a longer latent period. If a tracing be taken of the response of the soleus to a single induction shock it will be seen that the contraction is composed of two parts. This is shown in fig. 17, where the summit of the curve is seen to be distinctly double. The lever first rises quickly, though not as quickly as in the case of white muscle. It then forms a rounded summit usually a little higher than the first rise, the two being separated by a small notch. The relative heights, however, of the two components of the contraction are variable as is shown in the figures given.

On stimulating with the constant current a most curious condition is obtained. Sometimes a double contraction is obtained at make and at break resembling that which is obtained from induction shocks.

This is shown in fig. 18. Sometimes however the muscle enters at make into contraction which persists until break (fig. 19). The cause of this variation in the response of the normal red muscle I have not been able to determine. It may be an individual peculiarity, or it may

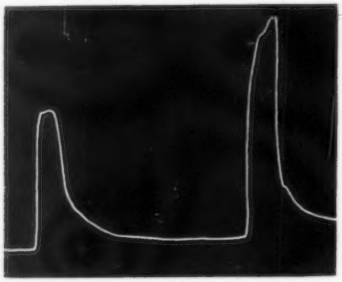


FIG. 17.—Normal soleus. Two induction shocks.

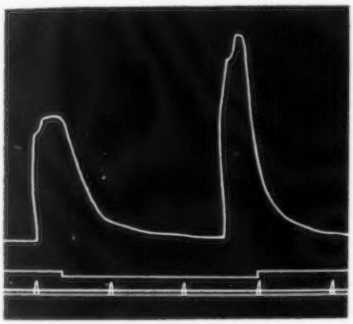


FIG. 18.—Normal soleus. Constant current. Bipolar stimulation. Time in seconds.

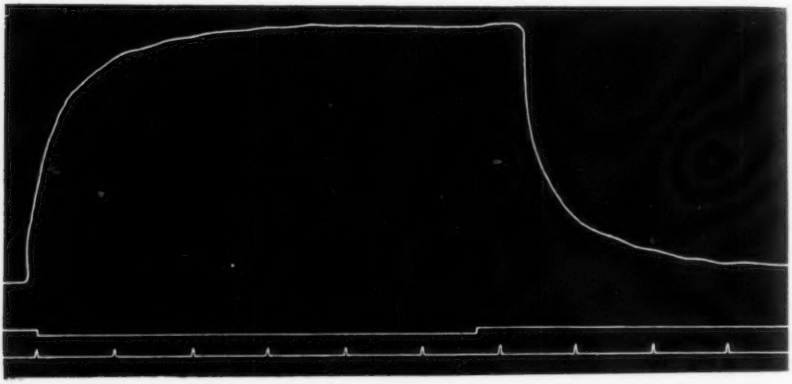
be due to the age of the rabbit. One thing is certain and that is that it is not a question of strength of stimulus. If the make and break of a constant current cause the double form of contraction, as in fig. 18, greatly increasing the strength of current will not convert this into a persistent contraction (fig. 20).

(8) *Effect of Load on Normal Soleus.*

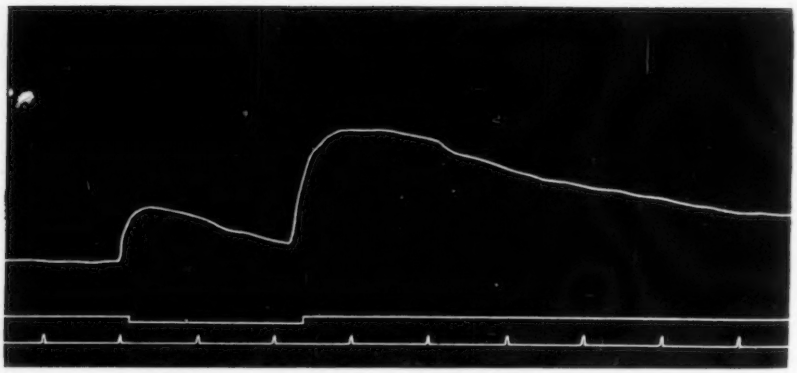
The effect of load is shown in fig. 21. It will be seen that as in the case of the degenerated white muscle increase of load diminishes the

height of contraction, but the persistent contraction is well maintained, even with 130 grm.

I have performed experiments in order to determine whether the resemblance between normal red and degenerated muscle can be pushed



a



b

FIG. 19.—Normal soleus. Bipolar stimulation with constant current. *a* = Ascending current. *b* = Descending current. Time in seconds; downstroke of the signal indicates make; upstroke of the signal indicates break.

further. Experimental difficulties have however prevented my arriving at any definite conclusion. The soleus takes origin by a small band from the posterior aspect of the tibia. On being traced downwards it is

found to be inserted for a considerable part of its length into a tendon which is common to it and to the inner head of the gastrocnemius. Through this attachment it acquires the greater part of its blood supply. When the muscle has been freed from its neighbours, an operation not

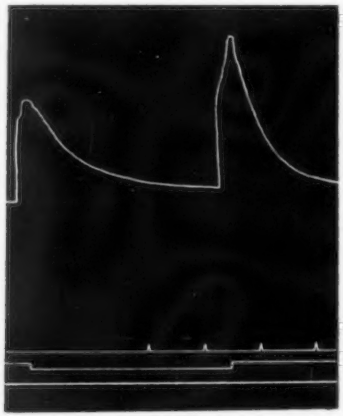


FIG. 20.—Soleus. Strong stimulation with constant current. There is a poor development of tonus.

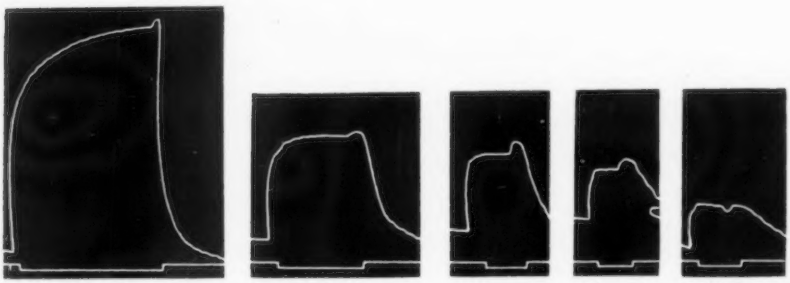


FIG. 21.—Normal soleus, showing effect of load. Bipolar stimulation : ascending current.

easy to perform without damaging its fibres, it is isolated from the circulation and soon loses its vitality. Moreover the extent to which the soleus is developed varies very greatly in different rabbits.

For the same reasons the study of the process of degeneration in this type of muscle has in my hands at any rate led to no definite

conclusion. In some cases the muscle has atrophied so quickly as to be almost incapable of contraction by the second week. Sometimes it has been possible to procure galvanotonus in the degenerated muscle when this condition was not present in its normal fellow of the opposite side.

(9) *Dual Theory of Contraction.*

The results so far obtained show that when white muscle degenerates, its electrical behaviour shows a striking resemblance to that sometimes shown by the undegenerated red muscle. Now the essential structural difference of the two varieties of muscle is as we have seen the greater amount of sarcoplasm in the red than in the white. Bottazzi [5] has argued that the two structural elements of muscle, the sarcostyle and sarcoplasm, are both capable of contraction, the former causing brisk, the latter maintained contraction. Joteyko [14] applies this theory to degenerated muscle, and assumes that the sarcostyle becomes inactive, the sluggish response being due to the sarcoplasm.

The process of reasoning which leads Joteyko to this conclusion is as follows: White muscle poor in sarcoplasm contracts briskly, red muscle rich in sarcoplasm contracts slowly—therefore the contraction of the former is sarcostylic, that of the latter is sarcoplasmic. Since red muscle degenerates more slowly than white, sarcoplasm degenerates more slowly than sarcostyle. Therefore when white muscle degenerates the excitability of the sarcostyle diminishes while that of the sarcoplasm is exalted. In my experience however there is not a particle of evidence that red muscle does degenerate more slowly than white. In most cases the reverse is true. I consider, too, that Joteyko is wrong in attributing the contraction of red muscle entirely to sarcoplasm on account of the greater abundance of this constituent in red muscle. If this were so, what is the function of the sarcostyle in this type of muscle?

It is true, I think, that the phenomena of degeneration can best be explained on Bottazzi's dual theory of muscular contraction, but not for the reasons put forward by Joteyko. Galvanotonus, as it occurs in the degenerating white muscle, is best explained on this hypothesis. There are present in muscle two contractile substances, sarcostyle and sarcoplasm. The former causes brisk contraction, the latter a more sluggish contraction, preceded by a longer latent period. In normal white muscle the sarcostyle alone responds, the sarcoplasm being relatively scanty. A single brisk contraction therefore occurs. A constant current applied

to a red muscle brings about as we have seen a double contraction, the first due to the sarcostyle, the second due to the sarcoplasm, the extent to which the second contraction occurs varying in different individuals according to the relative amount and excitability of sarcoplasm in the muscle. Presumably, when the sarcoplasm is well developed the muscle exhibits galvanotonus. The comparative slowness of the primary contraction of red muscle is the property of the sarcostyle. Sarcostyle responds to stimuli of momentary duration, such as induction shocks, while sarcoplasm is best stimulated by current of comparatively long duration. In a white muscle, when degeneration occurs, the excitability of the sarcostyle is diminished, hence the decreased response to the faradic current. At the same time the excitability of the sarcoplasm is increased, the muscle responding to the constant current by a double contraction and showing galvanotonus. The white muscle, therefore, in degeneration comes to resemble the normal red. There is indeed only one other conceivable explanation of the form of contraction obtained in the degenerated white muscle. It might be due to the process of degeneration affecting the individual fibres unequally. If this were the case the muscle at a certain period after section of the nerve should be composed of fibres in all stages of degeneration. The contraction would therefore simply be more prolonged, and would show no sharp differentiation into two parts.

That two contractile elements in the individual fibres are responsible for the changes seen in degeneration is further shown by the effect of fatigue. It has been seen that sarcoplasm is more readily fatigued than sarcostyle, but that sarcoplasm itself is fatigued as it is stimulated much more readily by current of long than by currents of short duration.

It has been shown by Parnas [21] that the postural tonus in the adductor muscle of Mollusca is associated with a gaseous metabolism, which is very little above that of the uncontracted state. Roaf [26] found that in decerebrate rigidity the amount of CO_2 formed is not appreciably greater than in paralysis due to curare. The metabolism involved in galvanotonus will be the subject of a further communication. We may note here the fact above shown that in the degenerated muscle whereas the power of maintaining a load when once it is lifted is well developed, yet after the real mechanical work of lifting the weight several times tonus is considerably impaired. In view of Sherrington's belief that postural tunus is sarcoplasmic the inability of sarcoplasm to perform mechanical work would seem to be in harmony with the results obtained by Parnas and by Roaf.

For the contractility of sarcoplasm there is abundant evidence in comparative morphology. The power of alteration of shape is present in the protoplasm of amœba, and in all cells of amœbic form. At a stage higher in animal development we find the appearance of fibrils specially developed for the production of quick movement only. At the same time the organism still retains the power of slow contraction, the fibrils not being used in the process. This was first shown by Lieberkühn in the stalk of Stentor: "If as often happens the animals shrink slowly together during several seconds, instead of contracting suddenly, the fibrils instead of being short, thick and straight at the maximum of contraction, will be distinctly wave-like, and not perceptibly thicker than in the ordinary extended state of the animal. . . . When after slowly retracting the animals are almost globular, there is still a possibility of contraction, every fibril suddenly becoming short, thick and straight." The slow contraction is here clearly due to protoplasm.

Lieberkühn then proceeds to show the effect obtained by exposing these animals to the constant current: "While the current is passing, the protozoa usually remains in a state of contraction, but when it is weak they relax completely after a time, even during the passage of the current."

A glance at fig. 10 shows the striking resemblance in the effect of the constant current on the lowest forms of animal life, and in the degenerated muscle.

In Stentor the fibrils were seen not to take part in the slow contraction. We may argue, therefore, that in the highly complex mammalian muscle the sarcostyle, the part corresponding to the fibrils, takes no part in slow or in maintained tonic contractions, these being performed by the less highly differentiated substance.

It is interesting in this connection to compare with the process of degeneration the action of veratrin. This drug, as is well known, greatly prolongs the contraction of a muscle due to an induction shock, though it cannot itself initiate a contraction. Bottazzi believes that this drug acts by exalting the excitability of the sarcoplasm. The fact that veratrin is said to enter into chemical combination with the muscle does not negative such a theory. The effect of veratrin diminishes after the first contraction has passed off, a fact which is readily explained by the greater liability of the sarcoplasm to fatigue which the study of the degenerated muscle has led us to believe. Further, veratrin increases the excitability of muscle, resembling again the early stage of degeneration. Lastly, if a muscle be fatigued first, and then the drug applied

the excitability is increased. In this case the part which was originally fatigued is the sarcostyle, the drug acting not upon this but upon the sarcoplasm which is not fatigued, since by ordinary methods of stimulation the latter is not affected. If, therefore, Bottazzi is correct in assuming that veratrin acts specifically on the sarcoplasm, the resemblance between the veratrinized and degenerated muscle is additional evidence that the change in degeneration involves a disturbance of the normal relationship of the excitability of sarcoplasm and sarcostyle.

(10) *Function of Red Muscle.*

We have found that red muscle behaves so very differently when stimulated that we may by a short digression inquire into the function of this type of muscle. Why further are red fibres intermingled with the white in the same muscles in some animals, while in others are individual muscles composed exclusively of one kind or the other? Starling [28] shows by a comparison of the pectoral muscles of the falcon, the goose and domestic fowl, that a large proportion of red fibres is associated with the power of long distance flight. He argues from this that red fibres are developed "to carry out long-continued and powerful contractions." But if we compare experimentally the weight-lifting power of the gastrocnemius and of the soleus we find no evidence that the latter muscle is better adapted to this purpose. Moreover, so great is the size of the gastrocnemius, compared with that of the soleus, in a well-developed rabbit that it is difficult to imagine that the brunt of the heavy mechanical work falls on the soleus. If quick but light movement only were carried out by the gastrocnemius a smaller muscle would suffice.

Comparing the action of the pectoral muscles of the falcon with the corresponding muscles in the domestic fowl we find another difference besides the power of prolonged flight. The fowl performs its hurried flutter from the ground to the top of the wall by a series of rapid contractions, in which the whole of the wing takes part. On the other hand, the falcon, as Dr. Anderson reminds me, in its graceful flight, keeps the proximal segments of the wings permanently extended by tonic contraction of the pectoral muscles, while the distal segments perform the movement necessary for propulsion through the air. The pectoral muscles then being used in a different manner in the two birds, we may suppose that the difference in the two kinds of muscle fibre is associated with a different type of contraction, the white muscles for rapid, the red for persistent contraction.

Sometimes the two varieties are associated with difference in rate of movement. In the skate, for instance, Ranvier [24] has shown that the fin is moved forcibly and quickly in one direction by means of a white muscle, and slowly in the opposite direction by means of a red muscle, propulsion in one direction being thus attained.

In the case of the rabbit the anatomical relations of the red muscles are not without significance. The three best developed muscles of this type are the soleus, the crureus and the deep head of the triceps. Now if these muscles all entered into contraction at once the animal would, so far as the elbow, knee and ankle are concerned, be in a condition resembling decerebrate rigidity. It would, therefore, appear as though red muscle is concerned with the maintenance of posture. This would bring the red muscle of the mammal into line with the red pectoral muscles of the large birds. This is no red muscle to dorsiflex the ankle, because the rabbit never requires to maintain this joint in the position of active dorsiflexion. Again, from the different rate of contraction which distinguishes these muscles experimentally we may infer a similar difference of function. The even function of the tibialis anticus is to dorsiflex the foot, so that it may clear the ground while the leg is being brought forward for the next thrust. This is essentially a quick action, one cannot imagine any contingency in which the animal would have to perform this movement slowly. In flexion of the foot on the other hand we may well imagine that as this is as it were the essential act of progression a greater variety in rate of movement is required. This is provided by the presence of the two types of muscle on this side of the joint.

Another anatomical difference is this. White muscles sometimes unite bones of adjacent segments; much more frequently, however, they pass over two or more joints. The hamstrings, for instance, are attached to the pelvis and to the tibia, the gastrocnemius connects the os calcaneum with the femur, while the long head of the triceps originates from the scapula, and is inserted into the olecranon. Red muscles, on the other hand, usually unite adjacent segments of the limb, the soleus passing from the tibia to the heel, the short head of the triceps from the humerus to the ulna, and the crureus from the femur to the patella. Connected with this is the fact that red muscles always lie deeply to the white. Since white muscles which pass over two joints may cause movement at either joint, it is clear that if the full effect is to be exerted at one joint then the other one must by some means be fixed. Such fixation may be a function of red muscle. For

example, the hamstrings can either move the femur backward or they can cause flexion at the knee. But it would be impossible to get movement purely at the hip unless the knee were first fixed, and this could conceivably be done by tonic contraction of the crureus.

One further difference is to be noted between these two varieties of muscle. In some muscles the place of origin and of insertion are of small area, all the fibres passing from one end to the other. In others the attachment at either end may be of considerable extent, with the result that the fibres are of varying length. Now we find that speaking generally the white muscles belong to the first class. In the case of these muscles which pass over two joints especially. The red muscles belong to the second class. In the rectus femoris, for instance, most of the fibres arise from the pelvis and are attached to the patella; there is very little tendon insertion. The subjacent crureus, however, arises from a considerable area involving almost the whole of the anterior aspect of the femur. Consequently the fibres vary very considerably in length.

Now Lucas [18] has shown that in the case of amphibian muscle the fibres individually obey the all-or-nothing law as in the case of the heart. That is to say grading of contraction is brought about, not by variation in the amount of shortening of each individual fibre, but by varying the number of fibres brought into play. If this rule holds also for mammalian muscle, concerning which we have as yet no information, we can readily imagine that for graded movement the arrangement found in the red crureus is far superior to that found in a muscle of parallel fibres such as the rectus femoris.

In higher animals such as man we find that the red and white fibres are found not in separate muscles but in each individual muscle. Associated with this is the higher power possessed by man of performing a movement of accurately graded intensity in almost every muscle.

The functions of red fibres would then appear to be: (1) to play a prominent part in maintaining posture; (2) to perform slow movement; (3) to perform movement of graded extent; (4) to act synergically with white muscles which act upon two joints by fixing one joint, thus enabling the white muscle to act solely upon the other. Red fibres do not appear to be well adapted to perform mechanical work of a heavy nature.

Summary of Chapter V.

(1) The reaction of degeneration in mixed human muscle cannot be attributed to a more rapid death of the white fibres.

(2) White muscle at a certain stage of degeneration resembles very closely normal red muscle in its electrical behaviour.

(3) This is adduced as an argument in support of the theory that the brisk twitch of normal muscle is a contraction of sarcostyle, while the slow contraction of degenerated muscle is due to sarcoplasmic activity, the sarcostyle degenerating more rapidly than the sarcoplasm.

(4) Morphological and pharmacological evidence has been given for the contractility of sarcoplasm.

Chapter VI.—Electrical Reactions in Conditions affecting Muscles Primarily.

We have seen that the process of degeneration is the result of damage to the motor nerve or nerve cell. Changes in the electrical behaviour of muscles also occur in those obscure conditions in which the seat of the lesion appears to be not the nerve but the muscle itself. At the present day three such diseases are recognized—myasthenia gravis, myotonia congenita and amyotonia congenita.

(1) *Myasthenia Gravis.*

Myasthenia gravis is distinguished clinically by the readiness with which fatigue occurs. Histologically the muscles involved and certain other organs are the seat of lymphorrhages. According to Farquhar Buzzard [6], electrical examination shows that with the faradic current the muscles are readily fatigued and soon cease to respond. If the constant current be applied to a muscle thus fatigued, a brisk normal contraction is the result. According to other observers the behaviour of the muscles to the two forms of current is variable; sometimes cases occur in which fatigue is produced by the constant more readily than by the interrupted current. It is extremely difficult to see how this can possibly be the case unless, which is quite possible, the interrupted current used in the examination was far stronger than the constant current.

Buzzard has suggested an explanation of his results based upon Bottazzi's dual theory. He supposes that the condition is due to a failure of the sarcoplasmic element in the muscle fibre. But this hypothesis would only hold good if the sarcoplasm were normally more resistant to fatigue than the sarcostyle. This seems to be disproved by the experiments above described. Further, the paralysis of the sarco-

plasm does not explain why the fibre retains its excitability to the constant current when fatigued apparently to exhaustion by the interrupted current. If the contraction obtained by the constant current

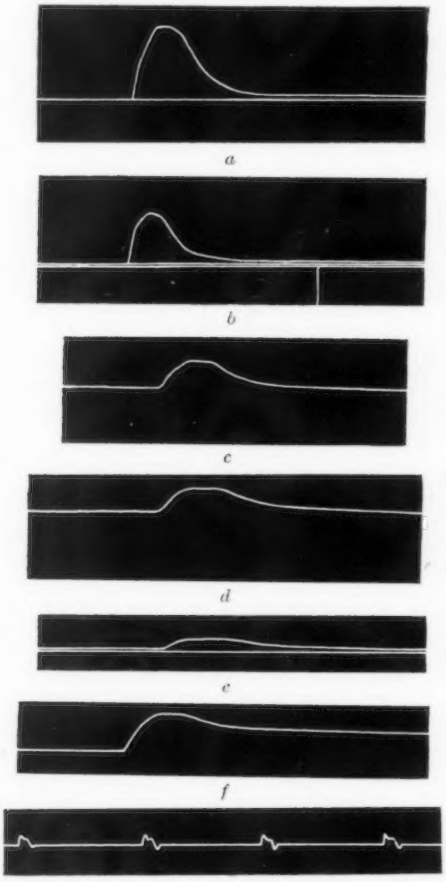


FIG. 22.—Muscle. Gastrocnemius of frog. *a*, Make of constant current. *b*, Single induction shock. *c*, *d*, and *e*, Induction shocks in stages of fatigue by the interrupted secondary current. *f*, Make of constant current immediately after *e*. Note the contraction is not as good as the initial one *a*, but is well marked and still brisk, as a comparison with the time record below ($\frac{1}{5}$ second) shows.

under these conditions were sluggish, resembling that which is obtained in the degenerated state, one might argue that in myasthenia gravis there is a pathological condition affecting the sarcostylic element,

rendering it abnormally liable to fatigue. But this is not the case. The problem before us is to explain why the muscle, though no longer responding to the faradic current responds in an apparently normal manner towards the constant current.

It is well known that the physiological efficacy of a current depends upon two factors—intensity and duration. A current of a certain strength of momentary duration may be totally ineffective, whereas a current of the same strength lasting for a longer period may cause a normal contraction. The two factors, intensity and duration, are related to one another inversely, the stronger the current the shorter the minimal period necessary to produce a physiological effect and vice versa. Now supposing a muscle be stimulated repeatedly by a current of certain strength and duration. Sooner or later the muscle, whether healthy or diseased, becomes fatigued and ceases to respond. But when this condition is attained the muscle can be made to contract by using a stronger stimulus. This can be done either by increasing the voltage of the current or by prolonging its duration. In this way a fair response can once more be obtained. This can be easily shown by the following experiment: The frog's gastrocnemius is dissected out and the distal end connected with a recording lever. It is first stimulated by the make of the constant current (*a*, fig. 22). It is then tetanized until the contraction obtained from a single induction shock is very feeble and sluggish (*b*, *c* and *d* show stages in the process). It is now once more stimulated with the constant current. A current stronger than that used before tetanization is required, but the resulting contraction, while not as sharp as the first contraction obtained (*a*), is nevertheless far better than the slow response obtained from the induced current. As observed by the eye it would undoubtedly be considered brisk.

We see therefore that the normal muscle behaves in the same way as the muscle in myasthenia gravis. The difference is one of degree, fatigue being induced far more rapidly in the latter than in the former. The explanation of the reactions in myasthenia gravis would therefore appear to be perfectly simple. The phenomena are due to an abnormal proneness to fatigue, which when it has once appeared affects the muscle in the same way as it does normal muscle. It is thus not necessary to assume that one histological element is affected more than the other.

(2) *Myotonia Congenita.*

In this condition all movements are first performed very slowly, and there is considerable difficulty in relaxing the muscles. With repetition

the contraction loses its sluggishness and eventually becomes as brisk as the normal. After a short rest however the sluggishness returns. Histologically there is a very striking increase in the size of the fibres, which may be from twice to four times as wide as normal. Transverse striation is but feebly marked and the nuclei of the sarcolemma are increased.

Electrical stimulation of the nerve trunk causes a prolonged contraction of the muscle. Similarly when the muscle itself is stimulated the resulting contraction is sluggish. In extreme cases it may last as long as half a minute. The muscle can be stimulated by K.C.C. and A.C.C. indifferently.

The clinical picture in this condition reminds one very forcibly of the action of veratrin, more especially in the prolonged relaxation. Possibly the hypothesis which Bottazzi brought forward to account for the action of the alkaloid may apply to this condition also, that is to say, there is an abnormal development in the amount and excitability of the sarcolemma. On stimulation it is the sarcolemma and not the sarcostyle which first responds—hence the sluggishness. The increasing briskness on repetition of the contraction is further evidence in support of this, for we have seen reason (Chapter V) to believe that the sarcoplasm is more quickly fatigued than the sarcostyle. As the excitability of the sarcoplasm decreases, the stimulus affects more and more the sarcostyle which is responsible for the brisk contraction ultimately obtained. That there is such a disturbance in two contractile elements is further indicated by the histological appearance of a muscle thus affected, the feebleness of the cross-striation indicating a comparatively poor development of the sarcostyle.

Chapter VII.—Wasting.

Wasting is so characteristic a feature in a muscle separated from its nerve-cells that it must be regarded as an essential part of the change which constitutes degeneration. It must be borne in mind, however, that this is not merely due to what is commonly called disuse, atrophy, though want of movement is no doubt responsible for some of the loss of substance involved. In the first place, atrophy due to disuse though in long-standing cases it may be extreme, never occurs so rapidly as the atrophy following nerve section. In a case of ulnar paralysis of about two months' history, the dorsal interossei will be found to be extremely wasted. But the small muscles of the hand, which are supplied by the

median, will present a striking contrast, even though the hand has been completely immobilized. If we use the term "disuse atrophy" in a broader sense, implying atrophy due not only to want of movement but also to want of tonus, then disuse may account for a still greater amount of the wasting which occurs. On the other hand, in paralysis due to causes other than nerve injury, however far advanced the wasting is, the electrical changes, characteristic of nerve injury, never develop. The atrophy of degeneration is therefore something more than the loss of substance due to want of exercise.

In the experiments described in Chapter V, I have weighed the muscles used—namely, the gastrocnemius, soleus and tibialis anticus—and have compared them with the corresponding normal muscles of the opposite limb. My first object was to find out whether there was any difference in the wasting of red and of white muscles, for it had been asserted that the former degenerated more slowly than the white. In the case of the soleus the muscle is so small in some animals, especially when degenerated, that it is not easy to weigh it with any accuracy. The difficulty is further increased by the peculiar insertion of the muscle, already noted above. The part of the tendo achillis into which it is inserted belongs chiefly to the gastrocnemius. In order to get the true weight of the soleus I have first freed the muscle from its tendon.

TABLE VI.—Showing Amount of Wasting.

No. of Rabbit	No. of days after section of nerve	Gastrocnemius			Soleus			Tibialis anticus		
		Normal	Degenerated	Percentage difference	Normal	Degenerated	Percentage difference	Normal	Degenerated	Percentage difference
1	3	8·83	7·64	13·5	0·87	0·71	18·4	3·12	2·82	9·6
2	8	10·97	7·30	33·5	1·60	0·77	33·0	3·82	3·39	11·25
3	10	8·10	7·33	10·2	1·30	0·74	43·0	4·02	3·91	2·7
4	14	10·00	6·90	31·0	1·20	0·85	29·2	..	..	..
5	14	11·34	8·50	25·0	1·49	1·30	13·0	..	..	..
6	15	16·70	10·65	36·0	1·60	1·32	17·5	5·75	4·80	16·5
7	21	10·90	6·50	40·0	0·7	0·9	..	3·27	3·50	..
8	30	15·35	6·80	55·4	1·22	0·37	69·7	5·60	3·50	37·5
9	34	8·84	4·04	54·3	0·72	0·67	6·9	4·12	2·23	45·9
10	53	10·25	6·07	40·0	0·97	0·52	46·4	4·05	2·75	32·1
11	58	13·07	5·22	59·8	1·45	0·47	67·6	5·30	2·75	48·1
12	70	6·75	4·95	26·7	0·73	0·27	63·0	2·10	1·75	16·7

The figures given in Table VI bring out one point very clearly, and that is the great variation to which wasting is liable. In one case

twenty-one days after nerve section the muscles on the paralysed side were actually heavier than the corresponding muscles. They may of course have been considerably heavier before the operation.

It has been customary among advocates of electrical treatment who wish to give a practical proof of the utility of artificial stimulation to demonstrate the increase in weight obtained in rabbits' muscles, unstimulated muscles being used as a control. Lewis Jones, for instance, used this method as a proof of the superiority of sinusoidal over the ordinary galvanic current. Recently Langley [16] has been unable to find any difference in weight as a result of stimulation. In these experiments the constant current and the condenser were used, the muscles being stimulated, some for short, others for prolonged periods daily. From my own experiments I am convinced that so great is the variation in wasting to which muscles are liable that experiments in which loss of weight is taken as the standard are of very little value. So far as my figures go they would appear to indicate that there is no great difference between the wasting in red and in white muscles. On the whole the former atrophy rather more quickly than the latter. If then the amount of wasting is any guide to the amount of degeneration, there is no evidence that red muscles degenerate less rapidly than white.

The results obtained, however, illustrate one point of a more positive character. It will be seen that the tibialis anticus in all cases wastes less than the gastrocnemius, in many cases the difference is considerable. Now it is well known that when the sciatic nerve is injured in man the anterior tibial group is affected to a greater extent than the muscles of the calf. Sherren [27] suggests that it is due to the more exposed position of the fifth lumbar root, and of the external portion of the great sciatic nerve in the thigh. But this does not explain the fact that the same difference is found in paralysis due to toxic neuritis. Hofman makes the improbable suggestion that the external popliteal nerve is more vulnerable, because it receives a smaller branch of the comes nervi ischadici. Now it has been well known to surgeons that so long as a paralysed muscle is stretched it will not recover. Neurologists and surgeons alike have drawn attention to the importance of keeping a paralysed muscle in a relaxed condition. This has again been very recently insisted upon by Robert Jones [13]. There seems no doubt, therefore, that stretching involves a considerable amount of damage. In slight injuries or affections of gradual onset, such as neuritis involving the sciatic nerves, the nerve may be affected as a whole, causing

paralysis of both muscle groups equally. As the patient continues to walk about, a new factor comes into play—namely, the weight of the foot. By this means the anterior tibial muscles are stretched while the gastrocnemii are released, hence the damage already done by the nerve injury is increased in the former, and diminished in the latter. If the patient lies in bed, the weight of the bedclothes on the toes acts in the same way unless specially guarded against. Drop-foot of toxic origin would therefore appear to be due, not to an affection of the external popliteal part of the sciatic, but to the stretching of the anterior tibial group after a generalized inflammation of the whole nerve. The same explanation accounts for the unequal fate of the two sets of muscles following wounds of the sciatic, a fact which has been noted both in the South African War and in the great war of the present day.

That this explanation is probably correct is confirmed by the relative wasting of the tibialis anticus and gastrocnemius in the rabbit. In this animal, as it feeds quietly in its cage, the hind legs are in a position of dorsiflexion, whether the sciatic nerve is cut or not. The gastrocnemius is thus subject to constant stretching, whereas the anterior tibial muscle is completely relaxed, hence the greater atrophy of the former than the latter.

The experimental results, therefore, strengthen the conviction of the overwhelming importance of keeping the muscles relaxed.

Summary of Chapter VII.

(1) The amount of wasting is so variable in the rabbit that any experiments in which loss of weight is adopted as a measure of degeneration are of little value.

(2) There is no great difference in rate of wasting between white and red muscles. In the majority of cases the red atrophied more than the white.

(3) In the rabbit the gastrocnemius wastes more than the tibialis anticus; this is explained by the stretching of the former and the relaxation of the latter in the position of dorsiflexion normal to these animals. In man the weight of the foot produces the reverse result, thus explaining the greater affection of the muscles of the front of the leg than of those of the calf.

Chapter VIII.—Conclusions.

(1) The normal muscle responds to the faradic current, whereas the degenerated muscle does not. To the galvanic current the normal response is brisk, the pathological is slow or vermicular.

(2) When the process of degeneration has lasted a month the degenerated muscle is less excitable than normal to the constant current.

(3) In normal muscle K.C.C.>A.C.C. so long as the testing electrode is placed on the motor point, this is not always possible in the case of small muscles. In the degenerated state the muscle tends to be indifferent to polarity; differences may exist in either direction, but these are small in comparison. In a normal muscle the K.C.C.: A.C.C. increases with increase of stimulus, in degeneration the relationship is not varied by increase of stimulus.

(4) Normal muscle is more excitable at its motor point than at any other part. Degenerated muscle is equally excitable at all points on its surface.

(5) In cases of peripheral nerve injury there is usually no doubt as to whether the muscle is degenerated or not. The question of practical importance is the extent of the injury which the nerve has suffered. To this question it must be admitted electrical reactions give but a limited answer, the chief reason being that the electrical response does not go pari passu with voluntary power. It cannot be too strongly insisted that the testing of the muscles is one only of several methods by which we can estimate the condition of the nerve. It is not the purpose of this thesis to discuss these methods, but we may enumerate them here. They are (i) the sensory loss, (ii) the trophic changes, that is to say the condition of the hair, nails and skin, and (iii) the degree of wasting. In cases of gunshot wounds of nerves, if the full reaction of degeneration is given by wasted paralysed muscles, if the sensory loss is full and trophic changes well developed, exploratory operation is advisable without delay provided the wound is aseptic. And this for two reasons: first, the small risk of the operation; second, the long period which must elapse before any objective change occurs if a waiting policy is taken. Cases in which the muscles give the reaction of degeneration where the wasting is slight, where the sensory loss is not complete, and when the trophic changes are not developed should be watched first for a couple of months or so. Injuries of this class usually recover without operative interference. These are the two

main types of nerve injury met with in the present war. Cases of an intermediate type, of course, occur. My own practice has been in such doubtful cases to advise operation. I may say that in a very large number of cases I have had reason to regret my decision in one only.

(6) This brings us to the question of subsidiary treatment by electricity and massage. Langley [16] has attempted to show by experimenting on rabbit's muscles that daily stimulation is useless. I have already shown that the results obtained in this way are inconclusive, and altogether beside the point. The results must be obtained from man. It is, of course, impossible to obtain any absolute proof by this method, but one has only to compare a limb which has been well cared for by constant stimulation with one which has been neglected, to be convinced of the value of electrical treatment in these cases. How the current acts is not certain. We may suppose that just as passive movement improves the condition of an affected joint by breaking down adhesions and exercising the muscles, so constant internal movement of the muscle itself maintains a condition of mobility in the sarcoplasm, and prevents fibrosis during the regeneration of the sarcostyle.

As to the form in which treatment should be administered I consider the constant current interrupted by a metronome to be the best. This should be done for at least fifteen to twenty minutes daily. Care must be taken that the current does not spread so as to stimulate the healthy muscles of the opposite side of the limb. If this is done the paralysed muscle is damaged by being stretched. For this reason the method of immersing the whole limb in an electric bath is strongly to be deprecated. The greater spread of current which occurs when the condenser is used makes this instrument less efficient as a means of treatment than the interrupted constant current. I have already insisted upon the importance of keeping the paralysed muscle permanently relaxed.

The clinical data of the above work were obtained from patients at the First Eastern General Hospital, Cambridge; the experimental portion at the Physiological Laboratory of the University. The expenses were defrayed by the Medical Research Committee, whose secretary, Dr. W. M. Fletcher, I have to thank for suggesting the experimental part. I am indebted also to Dr. H. K. Anderson, for the interest he has taken in the research, and to Mr. J. Barcroft for kindly facilitating work in a laboratory much depleted of assistants.

REFERENCES.

[1] Aeby. *Arch. f. Anat. u. Physiol.*, 1867, S. 688.
[2] Baierlacher. *Aerzt. Intelligenz-Blatt*, 1859, Bd. vi, S. 37.
[3] Bennett, A. H. "Electrodiagnosis." London, 1882.
[4] Biedermann. "Electrophysiology" (Trans. by F. Welby), London, 1896, vol. i, p. 273.
[5] Bottazzi. *Journ. of Physiol.*, 1897, vol. xxi, p. 1.
[6] Buzzard. *Brain*, 1905, vol. xxviii, p. 438.
[7] Dubois. *Rev. Internat. d'Electrotherapie.* Paris, August, 1900.
[8] Erb. *Deutsch. Arch. f. klin. Med.*, 1868, Bd. iv, S. 535.
[9] *Idem.* In Ziemssen's "Handb. der Spec. Path.," Bd. xi, 2. Hälfte; "Handb. der Krankheiten des Nervensystems," Leipzig, 1878.
[10] Fick. In Biedermann's "Electrophysiology," 1896, vol. i, p. 182.
[11] Hernaman-Johnson. *Lancet*, 1916, vol. i, p. 396.
[12] Jones, H. Lewis. "Medical Electricity," London, 1913, Sixth Edition, p. 362.
[13] Jones, R. *Brit. Med. Journ.*, 1916, vol. i, p. 641.
[14] Joteyko. "La Function musculaire," Paris, 1909.
[15] Langley. *Journ. of Physiol.*, 1905-06, vol. xxxiii, p. 374.
[16] *Idem. Ibid.*, 1915-16, vol. l; *Proc. Physiol. Soc.*, p. xxvi.
[17] Leicher, D. In Biedermann's "Electrophysiology," 1896, vol. i, p. 201.
[18] Lucas. *Journ. of Physiol.*, 1906-07, vol. xxxv, p. 103.
[19] *Idem. Ibid.*, 1909, vol. xxxviii, p. 113.
[20] Onimus et Legros. "Traité d'Electricité médicale," Second Edition, Paris, 1888.
[21] Parnas. *Pflüger's Archiv*, 1910, Bd. cxxxiv, S. 441.
[22] Paton and Findley. *Journ. of Physiol.*, 1916, vol. l, p. 193.
[23] Ranvier. "Traité Technique d'Histologie," Paris, 1875-88, livre ii, p. 508.
[24] *Idem.* "Leçons sur la Système musculaire," Paris, 1880.
[25] Remak. "Galvanotherapie der Nerven- und Muskelkrankheiten," Berlin, 1858.
[26] Roaf. *Quart. Journ. Exper. Physiol.*, 1912, vol. v, p. 31.
[27] Sherren. "Injuries of Nerves," London, 1908, pp. 30, 192.
[28] Starling. "Principles of Human Physiology," London, 1912, Second Edition, p. 201.
[29] Ziemssen und Weiss. *Deutsch. Arch. f. klin. Med.*, 1868, Bd. iv, S. 579.

ON DISTURBANCES OF THE LOCALIZATION AND DISCRIMINATION OF SENSATIONS IN CASES OF CEREBRAL LESIONS, AND ON THE POSSIBILITY OF RECOVERY OF THESE FUNCTIONS AFTER A PROCESS OF TRAINING.

(I) PARTIAL RECOVERY OF THE ACCURACY OF LOCALIZING TACTILE STIMULI, TOGETHER WITH A DISCUSSION OF THE FACTORS WHICH UNDERLIE THE PROCESS.

BY T. GRAHAM BROWN

AND

R. M. STEWART.

CONTENTS.

PAGE

I.—Introduction 348

II.—On Discrimination and Localization of Tactile Sensations 352

III.—Methods employed 371

(1) The Subject of the Experiment 371

(2) Methods of Examination 371

(3) The Method of Training used in the Experiment 376

(4) The Course of the Experiment 377

IV.—On the Conclusions which may be drawn from the First Part of this Experiment 378

V.—Analysis of the First Series of Tests during which an Attempt to educate Spots in Accuracy of Localization was made 398

(1) In General 398

(2) On the Records which were taken before Training commenced .. 399

(3) On the Records which were taken during the Training 405

(4) On the Records which were taken during the five days which immediately succeeded the First Period of Training 408

(5) On the Effect of the "Training" of the Palmar Set of Spots on the Right Index Finger—Comparison of the Records before and after it 413

(6) On the Question whether the Improvement in Accuracy of Localization after Training was confined to the Spots which were trained 426

(7) On the Question of the (apparent) general improvement of Localization of Tactile Stimuli which took place in the Untrained Fingers 437

VI.—Clinical Description of the Case 442

I.—Introduction.

It has been known for some time that recovery of function may take place after a lesion of the so-called "motor" area of the cerebral cortex in the higher mammals—although for a time after the infliction of the lesion there occurs paralysis of "voluntary" motion of the parts of the opposite side of the body which were served by the area destroyed.

The possibility that recovery of function may take place in a similar way after a destructive lesion of the so-called "sensory" area of the cerebral cortex is a question which has hitherto received little attention.

Such an investigation would be difficult (although perhaps not impossible) to perform in animals. It is obviously more easy to make in the case of man, but suitable cases are rarely encountered in civil practice. At the present time, however, much material for its prosecution is being provided by gunshot wounds of the cerebrum. Although we have had access to but few organic cerebral cases, of late we have been fortunate enough to have two such cases under our observation.

Although little or nothing is known of the manner of working of the mechanism in the cortex cerebri, it seems probable at any rate that the paths by which the impulses which subserve sensations are carried to the cortex are more complex than those which carry the so-called "motor" impulses for "voluntary" action away from it.

Head and his co-workers have shown that probably the afferent impulses which subserve the different forms of sensation conditioned by stimulation of end-organs in the skin and deeper structures are re-arranged in the spinal cord at or near the level of entry of the nerve-fibres which carry them. A second re-arrangement takes place at the level of the nuclei of the posterior columns of the cord in the medulla oblongata, while a third re-arrangement occurs at the optic thalamus.

In the spinal cord the afferent impulses which subserve the sensations of pain, of heat and of cold cross to the opposite side of that organ soon after their entry and then are carried upwards in secondary nerve-paths upon the side opposite to that from which they entered. The afferent impulses which subserve postural recognition and spatial discrimination run upwards upon the same side of the spinal cord as that of their entry. They travel along primary nerve-fibres in the posterior columns of the cord. The afferent impulses which subserve contact sensibility seem to travel upwards in a double path—partly on the same side and partly on the opposite side of the spinal cord to the side of entry. These impulses are evoked by touch and by pressure; this group carries with it the impulses which underlie the power of recognizing the situation of the stimulated spot.

At or above the level of the nuclei of the posterior columns of the cord the paths which carry the afferent impulses which subserve the sensations of pain, heat and cold run unaltered upon the opposite side of the brain stem to the optic thalamus; save only that there appears to be a certain degree of independence between the paths for the impulses

associated with these three sensations. The primary nerve-fibres which carry the impulses for postural recognition and spatial discrimination end in the nuclei of the posterior columns. Thence these impulses are carried upwards to the optic thalamus in two independent secondary paths—one for the impulses which subserve postural recognition and one for those which subserve spatial discrimination. The other uncrossed afferent paths now cross over to the side of the central nervous system opposite to that on which they entered.

According to the views of Head and Holmes [1], these various afferent paths—all now secondary—end in the optic thalamus where yet another redistribution takes place. From an examination of clinical material these observers believe that in the optic thalamus there is, besides the group or groups of nerve-cells at which all the afferent secondary nerve-fibres end, a mass of greymatter which they term the "essential organ of the optic thalamus." This, they suppose, forms the centre for certain fundamental elements of sensation. Its activity is mainly occupied with the affective side of sensation, and it is the "centre of consciousness" for certain elements of sensation—responding to all stimuli which are capable of evoking either pleasure and discomfort or consciousness of an internal change in state. The afferent impulses which subserve these sensations are presumably carried in short tertiary nerve-fibres from the nerve-cells at which the secondary nerve-fibres end to those in the essential organ of the thalamus.

Besides these short intra-thalamic tertiary nerve-paths there are, they suppose, longer tertiary nerve-fibres which carry on other afferent impulses from the nerve-cells at which all the secondary afferent fibres end in the thalamus to the cortex cerebri. These afferent impulses have been re-arranged in the optic thalamus and now run in five distinct paths—that is, in five paths, any one of which may be affected separately by a cerebral lesion. These five groups are: (1) the afferent impulses concerned with the recognition of posture and passive movement, and subserving the secondary faculty of discriminating weights on the unsupported hand; (2) those which subserve certain tactile elements (and also the discrimination of weights on the supported hand); (3) those which subserve spatial discrimination (the appreciation of two points applied simultaneously to two different points on the surface of the skin, and also the recognition of the size and shape of objects which are in contact with the skin); (4) those which subserve the power of localizing the situation of a stimulated spot of the skin, and that of the recognition of the double nature of two points applied

consecutively to too different spots on the skin; (5) those which subserve a scale of sensations with heat at the one end and cold at the other.

These observers believe that out of such materials the cortex cerebri fabricates the sensations for which it is responsible. They think that a great function of the cortex is the power of relating one sensation with another.

Now, if an object from the outside world comes into contact with a flat area of the skin the resultant stimuli engender afferent impulses which ascend in the central nervous system and finally subserve sensations of different kinds. These may be interpreted in consciousness in different manners. The relative hotness or coldness of the object, its roughness or smoothness, its size and shape, its weight and the part of the skin with which it is in contact, may all thus be perceived if we take a specific area of the skin, and apply tactile stimuli to spots within it; these stimuli may finally give rise to sensations which give information with regard to the locality of the touched spot within that area, or with regard to the doubleness of the touches if two different spots are touched either simultaneously or successively, and so on.

After certain cortical lesions the interpretation of these sensations may be greatly interfered with, and the description by the patient of the sensation (for instance, with regard to the localization upon the skin of a spot which has been touched) may be confused and wrong, or he may not be able to describe it at all.

The interesting question arises, whether in such a case it is possible to "educate" or "train"[1] the patient in such a manner that his replies to questions of this nature become accurate. Head and Holmes state that even twenty-three years after certain cortical lesions there may still be complete loss of the power of discrimination of two different points upon the affected side (compass-test), grave disturbance of the power of localization of tactile stimuli, and abolition of the power of appreciation of size, shape, and form in three dimensions, and of the power of recognition of familiar objects.

As far as we know, no attempt has hitherto been made in such a case to "re-educate" the lost faculties. The patient can hardly be expected to do this himself unless definitely directed to do so. For all practical

[1] We prefer the word "training" to signify a process by means of which such a lost cortical (or cerebral) function as that of localization might possibly become restituted. This word appears to us to have a more neutral meaning than "education" or "re-education," and to avoid committal to any theory with regard to a change which may possibly take place.

purposes his recognition of these attributes of objects is easier performed by sight (and, of course, by the unaffected hand), and there is little or no necessity for the "training" of recognition through the persisting sensations from the affected limb.

But if a definite effort is made is this "training" possible? If it is certain further questions of interest are raised. What are the factors which condition the appearance of improvement? Is it permanent or transient? Is it partial or complete? Again, suppose that it is possible to "re-educate" a lost faculty of the localization of tactile stimuli for certain definite spots on the skin, does the "re-education" affect the relative accuracy of the localization of tactile stimuli for other (let us say, neighbouring) spots which have not directly been "re-educated" for this faculty? Does a possible betterment of the localization of tactile stimuli for these specific spots which have been directly trained affect the accuracy of localization for other kinds of stimuli (that is, heat, cold, pain, &c., as well as other degrees of tactile stimuli than that used in the "re-education") for the same specific spots? And are other forms of sensation affected?

These problems are investigated in the experiment described in this paper. Before we attack them certain points in connection with the localization of cutaneous sensations may be considered.

II.—On Discrimination and Localization of Tactile Sensations.

When the normal subject of an experiment is so placed that he is unable to see a specific area of his skin—for instance, that of the palmar aspect of a finger—and a supra-liminal touch stimulus is then applied to a spot within it, he is able at once accurately to point to the corresponding spot on the hand of a model, although the only peripheral data which he receives are those of the touch stimulus itself.

The stimulus sets into action certain receptive end-organs in the spot of skin and underlying tissues. Thence afferent impulses pass into and up through the central nervous system until (probably) they effect activities in the cortex cerebri. There a complex physiological process occurs, the final resultant of which is the activation of certain "motor" mechanisms which in the end condition a very accurate movement of the hand which indicates the corresponding position of the touched spot upon the model. Parallel with the cortical changes there are changes in consciousness. The sensation conditioned by the stimulus is perceived; and, with it, various attributes of which the locus of the stimulus upon the surface of the body is one.

In the conditioning of the accuracy of this localization of the touched spot we may discern three factors.

In the first place, the sensations conditioned by the tactile stimulus must in some manner be different from those conditioned by all other tactile stimuli which are applied to other spots of the skin. This attribute of the sensation conditioned by tactile stimulus may be termed (for convenience here) its "character." It is clear that, if there was no such difference between the sensations which are conditioned by, or accompany, differently located tactile stimuli, accurate localization of any one of them would not be possible.

In the second place, the sensations conditioned by the tactile stimulus must in some manner be like those conditioned by all previous tactile stimuli which have been applied in the past to the spot which is now touched; and, moreover, it must also be like the sensations conditioned by other previous stimuli, such as those of pain, heat, or cold—for if one of these is applied to this spot it is also accurately localized there. This attribute of the sensations conditioned by the stimulus may be termed its "individuality." It is clear that if the sensations conditioned by different tactile stimuli which are applied at different times to the same spot have not a common "individuality" accurate localization of any one of them would not be possible, and the subject at one time might localize the stimulus in one place, and at another time in another place.[1]

These attributes of the sensation conditioned by a tactile stimulus are not, however, in themselves sufficient for the exact localization of a touch. The character of the touch may serve to distinguish it from all other touches which are applied to other spots, and its individuality may serve to liken it to all previous touches which have fallen on the same spot. The resultant localization might still be impossible or, although a constant one, it might be inaccurate. That is to say that the subject might localize a touch which is applied to a certain spot on the palmar surface of one of the fingers of his right hand at a dissimilar spot on the palm of the model, thinking that that spot corresponded in

[1] In this paper we use the term "location of the stimulus" to express the act of the observer in applying the stimulus to a certain spot on the body of the subject; or to express the locus of that stimulus as it appears to the observer. We use, on the other hand, the term "localization of the stimulus" (or "of the spot," or "of the sensation conditioned by the stimulus"—indifferently) to express the cerebral process whereby the subject finally indicates the position which he thinks that the touched spot occupies. We do not go into the question whether the subject localizes the stimulus, or the sensation conditioned by it, but use the two forms of expression indifferently.

position to the spot on his own finger which was touched. Each time that spot is touched on his own finger he may locate the touch on that one dissimilar spot on the palm of the model. In such a case the localization of the tactile stimulus would be constant, but it would be inaccurate.

A third attribute of the touch must therefore be its topographical position upon the surface of the body. This we may term its factor of "position."

These three hypothetical attributes of the touch—its character, its individuality, and its position—are not mere refinements of analysis. Their consideration has a very direct bearing upon the problem of errors of localization and of discrimination of tactile stimuli.

The effect of a certain lesion of the cortex cerebri may be to destroy the faculty of localization of a touch. That touch may still be perceived in consciousness as such, but the patient may be unable to refer it (or to refer it accurately) to the corresponding spot on the model (that is, to the "similar" spot on the model) if he is unable to see the spot which has been touched.

Now in such a case the subject may be unable to localize the touch at all. As in many of the cases cited by Head and Holmes, he may be "confused" when he is asked to point to the corresponding spot on the body of the model. He is completely ignorant of its location. There is no question here of the character, individuality, or position of the touch; the subject is, as it were, "cortically blind" to localization—just as "cortical blindness" is associated with certain lesions of the cerebrum.

But the subject (in other cases) might still think that he is able to localize the touch. In such a case he may make the attempt to localize it; but each time he is wrong in his localization. Touches which are applied at different times to one and the same spot of the subject's skin may be localized by him at different spots on the model: and different touches on the subject may, at different times, be localized upon the the same spot on the model. In such a case the faculty of localization may be said to be "impaired" or to be "abolished" for certain areas of the skin—but it is evident that the condition is a different one from that in which the subject is unable to make any attempt at localization of the stimuli. That is, of course, if it is certain that the subject is not merely guessing the positions of the stimuli.

In a case in which the subject thinks that he can localize his tactile stimuli the ordinary methods of examination yield results of little value for the analysis of his condition. It is not sufficient for the observer to

touch one of an infinite number of spots on the subject's hand, and to note simply that the spot is wrongly localized upon the model. Information is needed with regard to the wrongness of the answers. It is needed for data with regard to the question whether a touch which is applied to a certain specific spot is always referred to the same (wrong) position on the model; and whether that same position on the model is sometimes indicated as the corresponding location of touches which are applied to other spots on the subject.

In an ordinary test of the function of localization of touches the area investigated is usually restricted. That is to say, that the subject usually knows that the stimulus will be applied to a spot on a certain restricted area of the skin—for instance, to the skin of the hand, or to that of the arm, and so on. If a further restriction is made, and the subject is allowed to know that the stimulus will be applied to one of a definite small number of spots upon (let us say) his hand, the conditions of the experiment are such that information with regard to the manner of the wrongness of localization of touches may be obtainable.

Now, if the character of the sensations conditioned by tactile stimuli applied to our chosen spots is lost, the subject may refer the locus of a stimulus which falls upon one of them to a certain spot on the hand of the model. It is theoretically possible that he may think that he has made an accurate localization of it; but when a certain other spot is touched he may also refer the locus of that touch to the same spot on the model, and still think that the localization is accurate. In other words, he may think that the two different tactile stimuli are applied to the same spot, whereas, in fact, they are applied to different spots. If the character of the touches is lost for spots in a certain area of the skin it might be thought that the attributes of individuality and posture would be lost at the same time. This is, perhaps, not necessarily the case. Thus the touches applied to spots in that area might lose their attribute of character—so that they would seem all to have the same locus. The touches become similar, and at the same time the attribute of individuality of the different touches would merge in a common individuality. This is not the same as a loss of the attribute of individuality; we might say, perhaps, that difference of individuality is a function of the characters of sensations conditioned by differently located tactile stimuli. When the attribute of character is lost the attribute of individuality may become a common one for sensations conditioned by tactile stimuli applied to the spots in this area. In a similar manner, the attribute of position might possibly also persist

even after the loss of the attribute of character. Thus the differences of individuality of the sensations conditioned by tactile stimuli which are applied to spots in a certain area of the skin having been merged in a common individuality, the subject may still think that he can localize the touches. Here he has, as it were, a number of possible places upon which he can localize the touches which seem to him to be similar. One of these may be chosen (perhaps, by "chance"), but thereafter he would be expected to continue to localize all these touches on this one spot of the hand of the model—at any rate, during the course of one series of readings.

In such a case the first localization of a touch which is applied to the affected area may be a matter of chance. Thereafter, all other touches applied to different spots in this area will be referred to the same spot on the model. But if a limited number of spots are taken for examination in the affected area of skin; and if these spots are marked upon the hand of the model; and if the patient knows that the touches of which he is conscious must be localized upon one of these spots on the model or not at all; when the different spots on the patient are touched in an irregular order the patient will either localize the touches all on one of the spots on the model—or will indicate here and there that he is unable to localize a touch. If failures to localize are disregarded, the answers will tend to run in long similar series and (provided each of the spots on the patient is touched the same number of times in the series) the average error will be the same as that of hazard—it will be the same, that is, as if the answers were chosen by lot. The following example from actual experiment is similar to what might be expected to be obtained. Three spots only are examined. The numbers on the upper line are those attached to them: those on the lower line are the numbers of the corresponding spots on the model which were indicated by the patient as corresponding to the location of the touches. The interruptions in the series were not present in the experiment and are here interpolated in order to indicate the series of similar numbers in the answers:—

232 1 3231 3 1 2 3 2112313211 3232313 213212 132312 1
333 1 2222 3 1 2 3 2222222222 333333 222222 111111 2

Had the answers in the above experiment been determined solely by chance, or had the same answer been given throughout the whole series, the number of wrong answers would have been 28—a percentage error of 66·7. Actually it is 23—a percentage error of 54·8.

In the example given above, it will be observed that, although the answers tend to run in series of similar localizations of dissimilar touches, the same answer is not given constantly. Often, but not always, the first answer in a series is a correct one, and for a time thereafter all the touches are localized on the same spot of the model. It may be that in such a case the attribute of character of the tactile stimuli is not entirely abolished, but is impaired; or it may be that the attribute of individuality is impaired, while the attribute of character is abolished. It is probable that the attribute of individuality attached to tactile stimuli has an element of "memory" in it. If this is defective, it might happen that although the characters of the spots in the affected area of skin are abolished, and the touches which are applied to them seem to the subject to be similar, yet the apparent individuality of the merged character might vary from time to time in the experiment. All the different touches then might at one time be thought by the subject to have the individuality of one of them, and they might be referred by him to the locus of that stimulus; whilst at another time these same different touches might be thought by him to have the individuality of another of them, and would be localized on another position.

If the attribute of individuality is not merely impaired, but is actually abolished along with the destruction of the attribute of character, and if yet the attribute of posture remains, the touches would appear to the subject to be similar, and he would think that he could localize the apparently common touch; but he would be expected to localize it, now here and now there, in the different possible positions. In such a case the error in localization would still be that of hazard; but the answers would no longer tend to run in long similar series.

The following example from actual experiment (in which the test was one of recognition of three different two-dimensional shapes applied to the palm of the hand) is similar to the records which might be expected to be obtained:

3123231312123121232132132313213212313123123
3321233212232132123212312312312321233132233

Here, out of 42 instances, in which three different stimuli are given each 14 times, 28 wrong answers are given. This gives a percentage error of 66·7 — exactly that which would be expected to be given if the answers had been selected by hazard. But on only five occasions is the same answer given twice consecutively.

Therefore it may be said that if the attribute of character of

touches is lost, but the attributes of individuality and position remain unimpaired, it would be expected that the localization of the different touches would be wrong (the average error being that of hazard); but the different touches would tend to be localized all on the same spot. If, however, the attribute of individuality is lost or impaired along with that of character, it would be expected that the error of localization would be the same as before (that is, the error of hazard), but there would be a tendency to indiscriminate variation in the position to which the touches are referred. If the attribute of position is lost, the patient would be expected to be confused, and unable to localize the stimuli at all.

It is possible to suppose that the attribute of character may be unimpaired, while that of the individuality of the touches is abolished. In such a case one touch will appear to the subject to be different from another, but a touch will not appear to be the same each time it is applied to a certain spot. Therefore if a number of tactile stimuli are applied in an indifferent order to a restricted number of spots on the affected area of the skin, the subject of the experiment will rarely confuse two different successive touches, but he will refer a stimulus which at different times is applied to one and the same spot, now to one of the possible positions, now to another, and only occasionally to the right one. In other words, where the tactile stimuli are not applied twice in succession to the same spot the answers will not tend to run in long similar series; but they will, nevertheless, tend to be wrong, and the average error will be that of hazard. The following example from actual experiment is similar to what might be expected to be obtained in these circumstances :—

2312312313213121312323123231312123121232132123123123213131213123313123131321312313123323213132123123123213131213123313123131321312313123323213132123

Here the number of wrong answers is 23 out of a possible number 42—a percentage error of 54·8. The question "3" is put 14 times, and is given right on 5 occasions and wrong on 9—a percentage error of 64·3. Of the wrong answers "1" is given 5 times, and "2" is given 4 times; so that the percentages of the possible answers — "1" and "2" (wrong) and "3" (right) — are 35·7, 28·6, and 35·7: that is, as near as is possible with a small number of observations to the proportions of hazard. In the case of "1" and "2" in this experiment, this close approximation does not occur.

If the attribute of position of the touches is lost along with that of individuality, the subject would be expected to be unable to make an

attempt at localization, although he might still be able to recognize that there was a difference between touches which were applied to different spots on the affected area.

Finally, if the attribute of position is alone abolished, it is theoretically possible that the subject may be able to recognize differently located touches as in some way different, and a touch as in some way similar to previous touches of the same spot, and yet would still be unable to make a localization of these touches.

We may restate the above paragraphs by saying that for the proper localization of a touch we must probably assume that the sensation conditioned by the stimulus has three attributes apart from its quality. These are—its character, its individuality, and its position. By restricting the number of spots touched in a test, and the number of possible replies of the subject, we have a means of distinguishing between them. If the attribute of position is destroyed the subject should (theoretically) be unable to make a localization, and the error may be termed one of 100 per cent. If the attribute of character is alone destroyed the subject should tend to locate touches which are applied to different spots upon a single spot, so that the answers to series of tactile stimuli, no consecutive two of which are applied to the same spot, should run in series of identical replies. The percentage error should be that of hazard. If the attribute of individuality is alone destroyed the subject should tend to distinguish between consecutive dissimilar touches, but should tend to localize any one individual touch indifferently on any of the possible positions. The percentage error should still be that of hazard, but the answers to a series of dissimilar tactile stimuli, no consecutive two of which are the same, should run in series of which also no consecutive two are similar. If a series of tactile stimuli are applied to the same spot the answers should vary.

It must be noted that, on theoretical grounds, abolition of the attribute of character along with that of individuality should give the same results in the answers to a series of tactile stimuli, no consecutive two of which are the same, as abolition merely of the attribute of individuality. Is it possible to separate these two hypothetical conditions by test?

The investigation of the effects of touching two different spots either synchronously or consecutively with the compasses may possibly give such a test. If the attribute of character is abolished, when the points of the compass are applied simultaneously to two different spots the resultant sensation should, theoretically, be one of singleness. And

singleness should also result when a very short interval of time separates the application of the first point of the compass from that of the second. These effects should also, theoretically, be obtained when the attribute of individuality is lost as well as that of character. If the attribute of individuality alone is lost the simultaneous application of the two compass points should theoretically give a feeling of doubleness, as should also the asynchronous application of the points to two different spots. Thus, on purely theoretical grounds, it is to be supposed that where the attribute of individuality alone is lost the compass tests will differ from those obtained when the attributes of character and individuality are both lost. In the former case there will be no failure to recognize the doubleness of two touches when the compass points are applied either synchronously or successively to two different spots in the affected area; while in the latter case such a failure should occur, and apparent singleness should be the result.

If, as on theoretical grounds seems possible, the localization test serves to distinguish between conditions in which on the one hand the attribute of character only is abolished, and on the other hand either that of individuality alone or those of character and individuality together are lost; and if the compass tests (discrimination of two different tactile stimuli as double whether they are applied simultaneously or successively) serve to distinguish between conditions in which on the one hand either the attribute of character alone is abolished or the attributes of character and individuality are both destroyed, and on the other hand the attribute of individuality alone is lost: then a combination of the two tests should serve to distinguish between the three possible conditions—that in which the attribute of character alone is lost, that in which the attribute of individuality alone is lost, and that in which the attributes both of character and of individuality are lost.

In other words, for the compass test to be a complete one the answer of the subject should not only indicate that there is a "doubleness" about the stimuli, but it should also give data with regard to whether the doubleness is referred to two different spots on the skin or to one and the same spot. When the compass is applied in such a manner that its points fall synchronously upon two different spots and the touches are referred to by the subject as having "doubleness," we may assume that this is referred also to two different spots on the skin. But this is not necessarily the case if the compass points are applied successively with a short interval of time between them (let us say 0·5 sec.).

To meet this problem the following compass tests may be used. They require two compasses, and one arm of one of them is furnished with a small soft pad of leather sufficiently far removed from the point that it cannot come into contact with the skin. Sometimes two different spots are touched successively with a short interval of time between the application of the first and of the second point of the compasses; sometimes a single spot only is touched; and sometimes the point of the compass which is provided with the leather pad is touched on a single spot, and then immediately afterwards an additional touch is applied to the pad of leather by the second compass—the interval between the first and second touch here being the same as that between the application of the first and second point of the compass when two different spots are touched successively. The subject states that the touch is a double one applied to two different spots; that it is a single one; or that it is a double one applied to the same spot. In the second test sometimes one point of a compass is applied to a single spot: sometimes the two points of the compass are applied successively to two different spots; and sometimes two compass points (one of each compass) are applied successively very close together to almost the same spot. The subject again gives one of the three above answers.

The normal subject can distinguish between the different elements of these compass tests. If, however, the attribute of character is lost (without the abolition of the other attributes and without any other disturbance) the subject will, theoretically, be unable to distinguish between the successive touching of two different spots and the successive touching of nearly the same spot with the points of two different compasses. In both cases, theoretically, he may recognize the touches as double; but the synchronous touching of two different spots will seem to him to be single. Where the attribute of individuality is lost alone the subject should theoretically be able to distinguish correctly between the two points applied synchronously to two different spots; the two points applied successively to the two different spots; the points of two different compasses applied successively to nearly the same spot; and of course the single compass point applied to one spot. If the attributes both of character and of individuality are lost the results of the tests should theoretically be the same as in the case of loss of character only.

With loss of the attribute of position alone (without loss of the other attributes) normal results should theoretically be given to the different compass tests even when the patient is unable to make the attempt at localization.

In these theoretical considerations we have of course made the assumption that it is possible that one only of the attributes of the tactile sensation may be lost, or that one may escape from a destruction which overtakes the other two. If such a dissociation between destruction of the different attributes does not occur—that is to say, if they are so closely bound together that if one is destroyed or impaired the others are also destroyed or impaired—it is clear that no tests will serve to distinguish between them. Then the effects of all cortical lesions which destroy or impair the faculty of localization of tactile stimuli will give similar results—which may, indeed, differ in degree, but will not differ in kind.

It is interesting here to note the observations of Head and Holmes [1] on this point. They state that "in a certain proportion of cortical cases it does not matter whether the compasses are applied simultaneously, or whether an interval is allowed to elapse before the second point touches the skin. The power of recognizing the double nature of the stimulus is lost and no increase of the distance between the two points makes a constant or material difference to the accuracy of the answers. . . . Evidently he (the patient) has lost that faculty by which 'two-ness' is recognized, and it does not matter whether the points are applied simultaneously or successively. Now, whenever successive application of the two points is recognized, localization will be found to be intact. The power of appreciating two points applied successively is in reality the faculty of localizing two spots that have been stimulated one after the other. It is independent of the power of discriminating two stimuli which act upon the surface at the same moment." To this question we shall return in parts of this paper to be published later.

In this analysis of the faculties of localization and discrimination of tactile stimuli we have so far confined ourselves to what may be termed "negative" error. It is clear that there may be a difference between possible errors of localization in which the subject, even although he thinks his localization in each case to be correct, places a specific touch, now here and now there, indifferently upon the different positions in a restricted test, and possible errors of localization in which the subject constantly places a specific touch upon the same wrong position. Let us suppose that the test for accuracy of localization is one in which a small number of different spots is used, and that the tactile stimuli applied to them are given in an indifferent order in which no two consecutive stimuli are applied to the same spot. In the former case the

total average error of localization will be that of hazard, and if the answers to any one specific stimulus are examined it will be found that they vary amongst the possible answers—the individual average error of the answers to that stimulus still being that of hazard. But in the latter case the total average error might even be as high as 100 per cent., as might also be the individual average error of localization for each specific tactile stimulus used in the test. Thus let us suppose that three spots only are chosen for examination, and that the subject knows that there are three possible answers in the case of each tactile stimulus—only one of the answers being correct, that is, corresponding accurately to the locus of the stimulus. In the former case the subject will call stimulus "3" indifferently "1," "2," or "3"; and in a sufficiently large number of answers 33·3 per cent. will be given to each kind. That is to say, that of the whole series of answers 33·3 per cent. will be right; 66·7 per cent. will be wrong, and the wrong answers to each of the three stimuli will be equally divided between the two possibilities. But in the latter case the subject may always localize stimulus "1" at "2," stimulus "2" at "3," and stimulus "3" at "1." The average error will here be 100 per cent. for the whole series, and 100 per cent. for the answers to each specific stimulus, while the wrong answers to any one of the three stimuli will not be equally divided between the two possibilities.

In a case in which there is persistently a greater error than that of hazard it is clear that something more than the mere abolition of the faculty of localization (or of one of its factors) is present. There must be an actual distortion of it—an actual constant error, not an indifferent error. This error we may term a "positive" one.

On theoretical grounds we may assume for the moment that our hypothetical attribute of position may either be abolished or distorted by a cortical lesion. In the former case there should be no localization of a touch, even although its attributes of character and individuality are unimpaired. In the latter case localization may be present in these circumstances, but it may be inaccurate. That is to say, that the localization of a certain touch which has the attributes of character and individuality, but has a wrong attribute of position, may be done in an inaccurate manner, but the error will always be the same.

The method of testing localization for a restricted number of spots may bring out this type of error if it really occurs. The test in which the number of spots touched is not limited can hardly be expected to

do so, for there the error of hazard is about 100 per cent.; in such a case the only indication of this type of error is the personal opinion of the investigator that a certain tactile stimulus is always referred to a certain wrong position on the model. Records in which the number of different tactile stimuli are limited, as also are the possible answers, will give an error of hazard which is appreciably less than 100 per cent. (i.e., 66·7 per cent. where three different stimuli are used, 75 per cent. where four are used, and so on). If the average error in such records is constantly and appreciably greater than the error of hazard it may be inferred that the error is a "positive" one and not a "negative" one—to use the terms in the sense which we have already given to them. If such a record is analysed still further and the average individual error of localization for each tactile stimulus is obtained it may be found that these errors differ. Thus it might be that the total average error in the answers to the whole series of different stimuli is in fact about that of hazard, but that the individual average error in the answers to the stimuli which are applied to one of the spots is markedly greater than the error of hazard. A further analysis might indicate that in the case of the answers to this stimulus there is a preponderance of one specific wrong answer, and that the wrong answers to it are not evenly divided amongst the possible wrong answers

The following example from actual experiment brings out this point. It actually is a record of the answers to three different two-dimensional stimuli—flat objects of three different shapes applied to the palm of the affected hand:—

123231312321312132321213213232123132312123
132321213221312123121312211111112123223112

212313123123231213123123231212312132132313
333222223223311312222222222231211121211211

In this example the number of wrong answers is 51 out of a a total of 84 answers—an average error of about 60·7 per cent., the error of hazard being 66·7 per cent. The individual average error in the answers to "1" is about 35·7 per cent.; that for "2" is about 57·1 per cent.—smaller than the error of hazard; and that for "3" is about 89·3 per cent.—much greater than the hazard error. Each of the three different stimuli is given 28 times, and when the wrong answers to "2" are analysed it is found that "2" was called "1" 5 times, and was called "3" 11 times—there, therefore, being a marked disposition to call "2" "3" when it is wrongly named. Of the wrong answers to "3," 8 are called "1," and 17 are called "2." In this

case there, therefore, seems a definite confusion of the stimulus "2" with the stimulus "3" and the error is too great to be due to hazard. As a matter of fact the individual average error per cent. in the answers to the stimulus "3" on six consecutive days was 75, 85·7, 89·3, 82·1, 78·6, and 71·4—in each case above, and sometimes markedly above, the error of hazard, which here was 66·7 per cent.

What may be supposed to happen in our so-called "positive" error of localization is that the spot indicated by the subject as corresponding to the position of the touch which he perceives lies in some constant relation to the true corresponding position. As Head and Holmes point out, Russell and Horsley [1] state that this is the case in disturbances of localization by cortical lesions. The former observers, however, believe that their own failure to discover such a uniform tendency to erroneous localization is due to the avoidance of the "groping" method of testing localization—the method in which the subject (not seeing the spot stimulated) tries to touch with his other hand the spot to which the tactile stimulus has just been applied—and to the elimination of error due to defective recognition of the posture of his limb by the patient. They say, "We cannot insist too strongly that, when localization is defective from lesions of the cortex, the stimulus is not localized in some false direction, but the patient has a vague and uncertain idea amounting in some cases to complete ignorance of its position."

Another point in connection with our hypothetical attributes of character, individuality, and position may be noticed. The attribute of position has obviously attached to it (or has as a part of it) permanent memory. It is the relating of a tactile sensation, the character and individuality of which are presupposed, to a certain part or point in the subject's "schema" (to use Head's term) of the surface of his body. For the accuracy and constancy of the attribute of position of touches applied at different times to the same spot of the skin permanent memory would seem to be necessary. In a similar manner permanent memory would seem necessarily associated with the attribute of individuality—perhaps the two attributes are closely connected, at any rate as regards this permanent memory. But is permanent memory of necessity connected with the attribute of character?

Theoretically, at any rate, it is possible to suppose that this sort of permanent memory is not necessarily attached to the attribute of character. Thus it may be conceded that tactile stimuli applied to different spots on the skin condition activities in the cortical mechanism which are in some manner different. The parallel changes in

consciousness which accompany these changes in activity may also be different, and in short, the sensations conditioned by the different stimuli are recognized in consciousness to be different—if they are present in consciousness at the same time, and can thus immediately be compared the one with the other. But if the different stimuli are not applied synchronously it would at first sight appear that an element of memory is necessary for the bringing together of the sensations which they subtend and for their comparison in consciousness. This is, perhaps, not necessarily so.

If the sensations conditioned by tactile stimuli were of infinitely short duration in consciousness, the comparison of two asynchronously applied stimuli would appear to be inconceivable without a link of memory. But it is probable that the sensation conditioned by a tactile stimulus has a definite duration in consciousness—there is a sort of "after-discharge." One of us has shown that electrical stimulation of a point in the "motor" area of the cortex cerebri is followed by a state of facilitation during which the point remains more excitable than usual to the electrical stimulus; and that the duration of this state—which is probably that of the "facilitation" which has been postulated for cortical "motor" (and also "sensory") activities—is a comparatively short one, 10 min. to 20 min. for liminal stimuli. Now, if we suppose that the activity which underlies the sensation in consciousness of a tactile stimulus may persist, slowly dying, after the withdrawal of a stimulus, and if this continuance of activity is paralleled by a persistence of the change of consciousness, another stimulus applied to a different spot during the continuation of the "sensory after-discharge" would be paralleled by a change in consciousness which would synchronize with the dying after-effect of the first. In a manner the two sensations would be present synchronously for comparison in consciousness. But it must be noted here that the dying after-effect of the sensation in consciousness may well be likened to a sort of transient memory; and that the after-discharge and dying state of facilitation of the physiological mechanism ("motor") might in a sense be regarded as the physical counterpart of such transient memory. It is not, perhaps, altogether unwarranted to apply this idea of facilitation to the "sensory" mechanism, for it seems to be present in the mechanisms of the post-central convolutions which are usually looked upon as "sensory" in function.

This state of "facilitation" may possibly be one of the physiological factors which underlie the psychical attribute of "character" in the

sensations conditioned by tactile stimuli. The recognition of different touches simply as different apart from the recognition of their locus may be conditioned in part on the physiological side by the state of facilitation of the mechanism. Thus a second stimulus applied sufficiently soon after a first to the same receptors should give a greater response; stimuli of different intensity applied at different times to the same receptors are recognized to differ: if now stimuli of the same intensity are used and this is within the knowledge of the subject, stimuli applied sufficiently close together in time to different spots may be recognized as different. For if two stimuli of equal intensity are applied soon one after the other to the same spot the sensation conditioned by the second will appear to be more intense than that conditioned by the first. If two equal stimuli are applied one after the other and the sensations conditioned by them do not appear to the subject to be different in intensity, he may infer that the stimuli have been applied to different spots. This is, perhaps, too far-fetched, and at any rate such a factor could not be the sole condition of difference in character of touches, for it would be hard to explain by it the recognition of doubleness of contact when two different spots are touched simultaneously.

Before concluding this section of the paper we cannot refrain from a speculation which we think may lead into interesting paths of investigation.

The psychical process of the localization of sensations is, as it were, a process of apprehension, and this seems to be conditioned by three chief sets of factors—the sets of factors associated respectively with the "character," "individuality," and "position" of the sensation.

But the process of apprehension is associated with other than the more or less simple sensations which are conditioned by what for practical purposes may be regarded as one-dimensional tactile stimuli; that process is also associated with the compound tactile (and deep pressure) sensations which the stimuli engendered by two-dimensional objects condition—and with the compound tactile, deep pressure, and kinæsthetic sensations conditioned by the stimuli which three-dimensional objects engender.

And, again, the process of apprehension is not restricted to the sensations conditioned by the activities of the sense organs of the skin, subcutaneous tissues, muscles, tendons, and joints; it is thus also associated with the sensations conditioned by the activities of such organs of special sense as the retina and the cochlea.

The interesting question therefore arises whether these other processes (which are in some manner, perhaps, comparable to the process of localization) may be analysed in terms of the three same sets of factors—character, individuality, and position.

In the case of the apprehension of the sensations conditioned by two-dimensional objects pressed upon the skin we have already satisfied ourselves that this obtains; and we have a little evidence which suggests that the process of apprehension of printed words and letters may also be analysed in terms of these factors.

Thus, for the proper description by the subject of flat objects of different shapes (of which he is allowed to be aware only through the sense organs of the skin and subcutaneous tissues), he must be aware of the character of each object—its difference with regard to the others, the individuality; its sameness each time it is pressed upon the skin; and the position—that is, its relation to one of his "schemata." It must be noted here that in the process of localization the factor of "position" refers not at all to the place upon the body to which the stimulus has been applied, but to the place of the sensation within the subject's schema of the surface of the body. In this sense the word may also be used to denote one of the factors (or sets of factors) in the process of apprehension of two-dimensional objects; that is, it may be used to express the place of a certain two-dimensional object within the subject's schema of such things.

The heard or seen word is apprehended and recognized, and in the process of recognition there may, perhaps, be discerned the same three sets of factors. Thus, a certain word must in the first place be recognized as different from all others—there must be factors of character in the recognition of words; and it must be recognized as similar to itself each time it conditions sensations (either visual or auditory)—it must have individuality; while, finally, it must be related to the subject's schema of words—it must have position. With the written or printed letter or the unitary sounds in heard speech these three factors may, perhaps, be relatively simple; but with the heard or seen word they are obviously much more complex. This complexity is increased by the fact that certain similar words may have different "positions"; thus the printed word "smack" has its factors of character and individuality but many positions. Its meanings are various: "a small ship, a loud kiss, a sharp noise, a smart blow, a taste, a tincture, a small quantity," and so on—to take only its meanings as a substantive. Here the positions of the word in its recognition are probably a factor conditioned

not only by the seen word itself but by the context—its environment or "background." [It would, perhaps, be more correct to say that many different words have the same character and individuality, than to say that a certain word has many different positions.] In a similar manner heard words may have similar characters and individualities but different positions—such an example as "sow," "sew," is sufficient to demonstrate this.

Quite another form of complexity lies in the fact that words which have different characters and individualities may have the same position. Thus "gauge" and "measure" may have identically the same meaning. It is interesting to note that, in the case of two words which have many meanings, but only one common to both, the effect of the context is a predominant factor in the conditioning of the position.

Of course the schema of language is very much more complex than the schema of the loci of cutaneous sensations, but we suggest that there is much similarity between the two. The position of a word in the language schema (or schemata) may be related to all which we denote when we talk of the meaning of a word; but it must be observed that a single word may be related to many different schemata, each of which forms a part of the complex which we may term the schema of heard and seen language. The position of a word may be common to many schemata; and we may make a simple model of the relation between the position of a word and the different schemata to which it is related by figuring the schemata as geometrical planes, and the position of the word as a geometrical point. We may then figure the relation between a word and its schemata by supposing that the different planes intersect each other at a single point—the position of the word.

In the case of language the position of a word is common to at least two sensory complexes—the seen word and the heard word. Indeed, it must be common to more than these two, for the position of a word in the process of recognition must be common to the different forms in which the seen word may be presented (that is, the written word and its different forms, the printed word and its different forms, the word written or printed in different colours of ink, the word as recognized by touch, the word as written or printed in different alphabets; for instance, in shorthand or in the raised characters of the Braille system of printing for the blind—the corresponding word in various different languages, and so on).

The subject's schemata of different classes of sensations seem probably to be (as it were) catalogues of common elements in many different sensations. This is, perhaps, peculiarly so in the case of the language schemata, but it must not be forgotten that it also obtains in so comparatively simple a schema as that in which the cutaneous sensations are related together in the process of localization. Thus the sensations conditioned by tactile stimuli, by stimuli of heat and of cold, and by pain stimuli, all of which are applied to a given small area of the skin, are referred to a common locus in the process of localization. That is to say, that these sensations have a common factor of position in their localization. In other words, each of them is referred to the same part of the subject's schema of the surface of his body.

We would like to suggest that the method of experiment used in this investigation is eminently adapted for the proper examination of these different subjective schemata, and for the analysis of the factors in the apprehension of sensory complexes—either the relatively simple process of localization (which seems capable of analysis in terms of three or more sets of factors) or the relatively complex process of apprehension of heard and seen language.

That method—which we believe to be a new one—necessitates a subject in which there is some disorder of the process which is to be investigated. Given this condition, the method consists in the restriction of the number of different stimuli presented to the subject (be they tactile stimuli, the stimuli conditioned by two-dimensional objects, auditory stimuli, visual stimuli, and so on), and in a restriction the number of answers which the subject is allowed to give. This restriction of the answers is obtained by allowing the subject to know the number and the different forms of the stimuli which are to be presented to him, but not the order in which they will be given. Accurate records of the answers (whether they be right or wrong; and, if wrong, the nature of the reply) are taken, and are then finally analysed after a correct estimation of the percentage error of hazard. The answers to the stimuli are scrutinized in the first place with reference to their order in series. Attention is paid to the possible error of giving the same reply consecutively to consecutive stimuli of different sorts—this type of error denoting defect in the factor of character—and to the possible error of giving different consecutive replies to consecutive stimuli of the same sort—this type of error denoting defect in the factor of individuality. In the second place the replies to each individual sort of stimulus are scrutinized after determination of the

hazard proportion of each possible answer to the sort of stimulus which is being analysed. A reply which is given in a greater proportion of times than the hazard proportion may be termed a positive one, and where the reply is incorrect it may then be termed a positive error. The occurrence of positive error seems definitely to point to a distortion of the position factor in the apprehension of the sensation conditioned by the stimulus which is being analysed.

This method of investigation, which may be suggested as one suitable for use in all cases of disturbance of the process of apprehension, may be termed the method of restricted test analysed in terms of positive error by reference to the error of hazard.

III.—Methods employed.

(1) *The Subject of the Experiment.*

J. H. L., born in 1886, wounded on October 26, 1914, by a rifle bullet through the left side of the head.

The wound of entry is represented by a small round hole in the skull, and about 1·5 cm. in diameter. Its nearest point to the central line lies about 5 cm. from it (on the left side of the skull), and the distance between the nasion and that point on the central line of the surface of the head which is cut at right angles by a line passing through this wound is about 12 cm.

In a similar manner the wound of exit lies (at right angles to the central line) 3 cm. to the left, a point on the central line—the distance between the nasion and that point being about 29 cm.

There is a trephine opening in front of the wound of exit. Its longest axis is parallel to the central line of the head, and is about 7 cm. long. Its most anterior point lies about 3 cm. to the left of a point on the central line which is about 22 cm. posterior to the nasion; its breadth is about 4 cm.

A description of the case is given in a later section of this paper. The experiment was begun on January 7, 1916, more than fourteen months after the infliction of the wound.

(2) *Methods of Examination.*

In this experiment the accuracy of the subject's localization of tactile stimuli of different intensities, of painful stimuli, and of stimuli of heat and of cold, was registered at different times. So, too, was the

accuracy of his responses (as regards doubleness or singleness) in the compass test, and the accuracy of his recognition of three differently shaped flat objects of indifferent temperature placed in contact with the surface of the palm.

In testing the accuracy of localization of tactile or other stimuli, we made use of the Henri method as modified by Head and Holmes. Stimuli were applied either to the dorsal or to the palmar aspect of the hands (the right hand being that which was affected), the arm first being passed through the opening of a screen which prevented the subject from seeing the spots which were touched. A model held his corresponding hand in the same posture as that of the subject. This hand was within the subject's field of vision, and he indicated upon it with a pointer (held in the other hand) the position which he thought to correspond to the location of a tactile stimulus which had just been applied to his own hand. The normal hand was withdrawn from behind the screen (in order to indicate the localization when control observations were made upon it).

For the purposes of our experiment, and for ease of registration, we restricted the loci of the stimuli to nine different specific spots on each of the second, third, fourth, and fifth digits of either hand. Of these nine spots, in each case three were situated on the dorsal aspect of the finger (see fig. 1), and were so placed that a spot lay on the skin about midway between the two extremities of each of the three phalanges. In the case of each finger six different spots were situated upon the palmar aspect. Three of these spots were placed, as in the case of those on the dorsal aspect, one about midway between the two extremities of each of the phalanges (see fig. 2); the remaining three spots (see also fig. 2) were placed on the folds of the skin near the metacarpal-phalangeal, and the two inter-phalangeal joints respectively—that is to say, about midway between pairs of the other spots. All the spots lay upon that dorso-ventral plane which passed through the long axis of the finger.

This gave us three sets of three different spots each, and we numbered the different spots in each set "1," "2," and "3" in their order from the extremity of the limb towards the trunk. The spots were permanently marked with silver nitrate; and spots corresponding to them were marked upon the fingers of the model.

In an experiment a stimulus was applied to a certain spot and the subject then (a) failed to give any response (this failure being recorded), or (b) tapped the end of his pointer upon the table to indicate that he

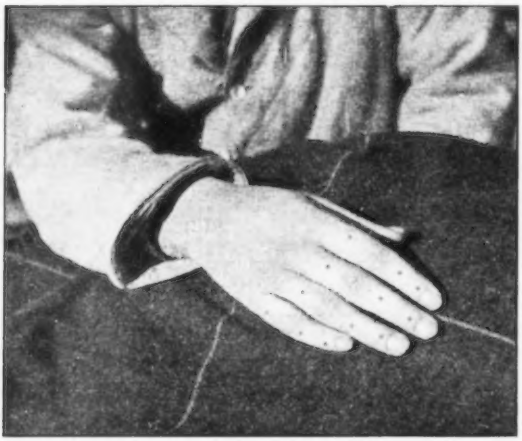


FIG. 1.—Photograph of the dorsal aspect of the subject's right hand. The three spots on each finger ("dorsal sets of spots") are shown, having been marked with ordinary ink for the purpose of the photograph.

FIG. 2.—Photograph of the palmar aspect of the subject's right hand. The six spots on each finger ("ordinary" and "untrained" sets) are shown with the exception of the distal spots (spots "1" of the "ordinary sets") on the index and ring fingers, which are covered by the observer's finger-tips. On the middle finger the spots from left to right (that is, distal to proximal) are "spot 1" ("ordinary set"); "spot 1" ("untrained set"); "spot 2" ("ordinary"); "spot 2" ("untrained"); "spot 3" ("ordinary"); "spot 3" ("untrained"). The black patch on the palm was the mark used in training experiments on discrimination of two-dimensional objects not here described.

was unable to make a localization of the touch which he yet perceived, or (c) touched with his pointer the marked spot upon the model which he thought corresponded in position with that to which the tactile stimulus was applied upon his own hand. If no localization was made (although the subject was aware of the stimulus) a mark was placed upon the record under the corresponding number of the spot which was stimulated, and that stimulus was not repeated. When no response was given—the touch not being perceived—the stimulus was repeated. If a localization was made the corresponding number which we attached to the spot indicated on the model was written below the number of the spot touched on the subject. "Dilemma" could usually be detected by observing the movements of the pointer, and when it occurred was registered on the records. The examination was conducted in silence, and throughout the whole duration of the experiment the subject was never allowed to see a record, nor on any occasion was he allowed to know whether his reply had been right or wrong. Owing to the slight contracture of the affected fingers one of the observers held the finger that was being examined in a posture of extension.

In the first part of the experiment we gave the stimuli to the different spots in a series, the order of the elements of which ("1," "2," and "3") was selected by lot. In consequence of this there were slightly unequal numbers of the three different spots in a series, and sometimes a certain spot would occur several times in immediate succession. In the succeeding parts of the experiment (not here published), we used a new set of records in which, while the order of the spots was selected by lot, no two consecutive ones were the same and in which equal numbers of each spot were present in each record.

We usually examined the accuracy of localization of touches for one of the sets of three spots on each of the fingers separately—the subject being allowed to know which finger was being touched in any one test. At first we used records in which twenty-five consecutive tactile stimuli were applied to the three different spots (an average of about 8·3 stimuli to each spot). Later we increased the number of stimuli in a series to forty-two, of which exactly fourteen were applied to each of the three spots.

The stimuli were applied at the rate of about one every five seconds, and intervals of one minute each were allowed to elapse between the series of stimuli which were applied to different sets of spots.

The sets of spots on the different fingers were examined in a constant order in the ordinary daily tests. First, two palmar sets—one on each

of the third and fourth digits of the normal left hand. Then a palmar set on the second, third, fourth, and fifth right digits in that order. Then the dorsal sets of spots on these six fingers in the same order as before. Finally, for purposes of control, the palmar set of spots on one of the normal fingers was again examined, and occasionally the palmar set of spots on the second right digit.

For purposes of control, and to obtain additional data, from time to time we did this test without allowing the subject to know which finger was being touched, but allowed him to know the position of the twelve spots which were to be touched upon the four fingers. In this test (as a rule) no one finger was touched twice consecutively, and the order in which the fingers were touched was otherwise selected by lot—equal numbers of stimuli, however, being applied to each finger in the test. In this test usually 84 stimuli were applied (sometimes 168) in consecutive series of 21 each, with intervals of one minute between the series.

The tactile stimuli used were given by pressing hairs, each of which exerted a constant pressure when force sufficient to bend it was employed (the hairs were of different stiffnesses in different tests) on the selected spots; painful stimuli by stabbing the spot with the point of a needle; heat and cold by applying a hot or cold flat metal surface (*circa* 5 mm. in diameter) so gently against the spot that it gave the least possible recognizable sensation of touch. We also on one occasion gave tactile stimuli by pressing the open end of the cap of a fountain-pen against the skin in such a manner that it encircled, but did not touch, the spots used in the other experiments. In the first series of records we used as a constant stimulus on consecutive days a hair of stiffness just insufficient to give a painful sensation when applied to a spot on the normal palm; in the following series of experiments we used a small apparatus for giving constant pressure-touch stimuli. This, which we made ourselves, consisted in a rod (the end being a bad conductor of heat) which worked in a hollow holder against an elastic band.

The normal error of localization of tactile stimuli applied with these instruments which we used in the day to day tests is, in the normal subject, about 0·0 per cent. on the palmar aspect when the test is restricted to a single finger. When it is not so restricted to a single finger, but when the possible spots on each finger are restricted to three, the normal error for the palmar aspect of the fingers seems to be about 8 per cent. The tactile stimuli are rarely localized on the

wrong finger (when they are the finger touched is usually the third or fourth digit, and the stimulus is referred to the fourth or third) and the error usually consists in wrongly placing a stimulus upon the right finger. The spot thus wrongly localized is usually "spot 3"—i.e., that of the three nearest to the trunk, less often "2," and seldom or never "1"—that situated on the pulp of the terminal phalanx. The error is usually made by localizing "3" on "2"; less often by localizing "2" on "3"; seldom or never by localizing either "2" or "3" upon "1." We have never, on our own persons, seen an error in which a tactile stimulus delivered with these instruments was localized wrongly both in regard to the finger and to the position of the spot on the finger.

(3) *The Method of Training used in the Experiment.*

The general aim which we had before us was the investigation of the possibility that a lost "sensory" function, probably of the cerebral cortex, can be regained—just as recovery of "voluntary" movement may occur in monkeys and higher apes after a cortical lesion of the so-called "motor" area. Although such a recovery of a lost "sensory" function has not yet been observed to occur "spontaneously," it is possible that it might do so under the influence of training. Tests of the accuracy of localization, &c., of tactile stimuli before and after such an attempt at training may yield evidence as to its effects.

In applying the training a single finger was selected and it only was trained at that time. Not only this. The training was applied only to one of the three sets of three spots on that finger—the other two sets remaining untrained. The three spots which were trained were, in every case, those situated about the middle points of the pads of the fingers which lie over the middle of the palmar aspects of the three phalanges.

In this manner we were able to compare the accuracy of localization of stimuli applied to the "trained" spots after the training with that of corresponding but untrained spots on the other fingers; with that of the untrained spots on the back of the trained finger; and with that of untrained spots on the palmar aspect of the trained finger (these untrained spots lying on the folds in the skin near the metacarpal-phalangeal and the two interphalangeal joints).

The training was performed in the following manner: An ordinary localization test having been done for the spots which were to be trained, the subject was allowed a short interval of rest. Then he was told

that spot "1" was going to be touched and that he was to concentrate as much as possible upon his experiences during the stimulation—trying to realize the peculiarities of the stimulus, &c. That spot was then slowly stimulated 10 or 20 times in succession with the ordinary instrument—and he was allowed to see each alternate touch being given by the silent removal of the screen. In the same way 10 (or 20) successive touches were applied to each of the other spots—"2" and "3." After a short interval of rest the subject was allowed to look at his finger while 50 successive touches were applied in an indifferent order to the three different spots. Then, after another interval of rest, the ordinary test of the accuracy of localization of tactile stimuli on these same three spots was again done—the subject now not being allowed to see his hand, and having to point to the corresponding spots on the hand of the model. During the training the subject was at one time told to concentrate his mind especially upon the similarity of the stimuli which were then being applied consecutively to the same spot. At another time he was told to concentrate upon the dissimilarities of stimuli which were being applied consecutively to different spots.

This training was usually done in the evenings of a series of consecutive days; sometimes it was performed repeatedly (at two-hour intervals) on the same day. During and after a period of training the ordinary tests were applied in the morning.

(4) *The Course of the Experiments.*

In our first series of tests, on 13 consecutive days we investigated the accuracy to localization of the spots on the palmar and dorsal aspects of each finger of the right (affected) hand of the subject, as well as the corresponding spots on the palmar and dorsal aspects of two of the fingers of the left hand. The stimulus used was the same throughout—that given by pressing a hair against the spot (the hair being a fairly stiff one, but not one which gave a painful stimulus on the normal hand). In addition we used the test in which the stimulus was applied to spots on any of the fingers.

On the fifth, sixth, and seventh days of the series the three palmar spots of the right index finger were trained. The accuracy of localization for tactile stimuli before the training was on this finger intermediate between that of the most defective and that of the least defective finger—but nearer to the former than to the latter. On the seventh day of the series the accuracy of localization on the right index finger was tested (after the training in the morning) every hour from 11 o'clock

in the morning to 9 o'clock at night. Thereafter the tests were continued as before; but on the ninth day of the series the accuracy of localization on the right index finger was tested thrice during the day at four-hour intervals; on the tenth day of the series it was tested four times during the day at three-hour intervals; and on the twelfth day of the series it was tested seventeen times during the day at half-hour intervals. In addition to this, on the tenth, twelfth and thirteenth days of the series the accuracy of localization of tactile stimuli was tested for the set of three untrained spots on the palmar aspect of the index finger (that is, the spots situated near the three joints)—and for the corresponding spots on the other fingers. While on the thirteenth day of the series the training of the spots of the palmar aspect of the right index finger was performed again, on this occasion the training being done four times during the day at two-hour intervals.

On the fifteenth day of the test (three days after the fourteenth) we asked one of our colleagues (Dr. W. H. R. Rivers) to make an examination of the affected hand before making him aware of the nature of our experiments. He came to the conclusion that there were gross errors of localization of tactile stimuli which were applied to the palmar and dorsal aspects of the right hand.

IV.—On the Conclusions which may be drawn from the First Part of this Experiment.

For the easier reading of this paper, we think it best to give here the conclusions which, we believe, may legitimately be drawn from the results obtained in that part of the experiment with which we are dealing—reserving the more detailed analysis of this part of the experiment to the following section of the paper.

It must be clearly understood in the first place that we have no definite knowledge of the situation, nor of the extent, of the cerebral injury—save only that in part that injury is situated in the parietal lobe of the left cerebral hemisphere. We have also no definite knowledge of any changes which may have taken place in the cerebrum during the course of the experiment, but we think that, as we started the experiment fourteen months after the infliction of the wound, any possible process of recovery should by that time have ceased.

The part of the experiment which we consider here is a small portion of the whole. We give the facts as they were; and our conclu-

sions are drawn only from the facts here given, and they are drawn irrespective of the facts obtained later in the experiment—save only that we know that a similar improvement of the accuracy of localization of tactile stimuli occurred when we selected another finger for training.

Now with regard to the state of the subject at the commencement of the experiment:—

The man was unable accurately to localize tactile stimuli which were applied (amongst other places) to the palm of the right hand and to the palmar aspects of the right fingers; the defect of localization was, in fact, gross. He was not, however, unable to attempt a localization. In fact, when a spot in that region was touched, the subject usually made a localization—even although that localization was often a wrong one. He, occasionally, was unable to make the localization at all when the test was limited to a certain small number of spots, but this inability lasted only for the first eight days of the experiment and the stimuli which he was unable to localize were only about 10 per cent. of the whole. Dilemma in the reactions of localization also occurred, but this also decreased as the experiment proceeded. Neither dilemma nor confusion occurred when the test was an unrestricted one—that is, the ordinary clinical method.

A striking characteristic in some of the cases cited by Head and Holmes was that the subject was frequently unable to make the localizations of tactile stimuli. In the present case there is no such inability—but a large proportion of incorrect localizations is made. Recently, we have had the good fortune to be able to investigate another similar case. In this second instance the error of localization is very large; but there is an almost complete absence of dilemma, and the proportion of instances in which localization cannot be made is small. In this second case a remarkable feature is the apparent certainty with which the subject makes the localizations. So great was this, that at first we suspected that he was making them by hazard. But the records show that this probably is not the case. We may conclude that in the subject of the experiments here described the effect of the lesion has not been to wipe out the function of localization of tactile stimuli for a certain part of the body, but only to destroy or to distort certain elements in the complete function.

In the complete process of localization of tactile stimuli we may distinguish three elements or attributes (as regards localization) of the sensation conditioned by each stimulus. These are: (1) the element of "character" (whereby the sensations conditioned by differently located

tactile stimuli are recognized as in some manner different from each other); (2) the element of "individuality" (whereby the sensations conditioned by tactile stimuli which are applied at different times to the same spot on the skin are recognized in some manner to be similar to each other); and (3) the element of "position" (whereby the sensation conditioned by a certain tactile stimulus is recognized as in some manner connected with a definite point in the subject's schema of the surface of his body). As in this experiment the spots which were touched lay upon the fingers of the right hand we may recognize at least two factors in the element of position. The first of these is the place of the touched spot upon the finger. But as there were four fingers all very much alike—that is, four different possible places for each spot—there must be a factor of "finger" in the position of the spot, a factor whereby the subject recognizes that in some manner the tactile stimulus has been applied to a spot on a certain finger. The elements of character and individuality are possibly more fundamental than the elements of position. The latter, perhaps, are those which relate the sensation conditioned by a specific tactile stimulus (with its character and its individuality) to the schema of the surface of the body.

As it is obvious that if the element of "position" in the localization of tactile stimuli is destroyed, the subject should be unable to make any localization of them, and as in this experiment such localizations were made (although often incorrectly), we may conclude that at any rate the element of position was not abolished. But it is yet possible that in our case the elements of position were distorted, although not eliminated. Our results definitely point to this conclusion.

Thus, in the first place, the error of localizing stimuli, which were applied to certain of our chosen spots, was greater than the error of hazard. Such an error may be termed (for our purposes) a "positive" one; and its occurrence clearly indicates that in these instances there was not a mere elimination of an element in localization, but a distortion of it. Thus the tactile stimuli, which were applied to a certain spot (e.g., spot "3") on a certain finger might be definitely thought by the subject to have been applied to another spot (e.g., "2") upon the same finger; and every time (or nearly every time) a tactile stimulus was applied to that first spot, it might be localized by the subject upon the second. In such a case what is at fault is the factor of "place," or "position," within a certain schematic field, of the sensation conditioned by the tactile stimulus, and the error is a definite one.

In the second place, the error of localizing a touch upon the correct

finger (that is, within the correct cutaneous field) was in some cases greater than the error of hazard. This shows that here also there was no mere partial elimination of a factor in the element of position, but a definite distortion of that factor. The subject definitely thought that a large proportion of the tactile stimuli which were applied to spots on a certain finger were applied to spots on a certain other finger.

It is quite possible that such distortions of the elements of position in the localization of tactile stimuli might be sufficient to account for the badness of localization in such cases as that under consideration, even if the elements of character and individuality in the tactile stimuli were perfect. But in this case distortion of the elements of position can hardly have been the sole condition of the badness of localization.

Thus the subject at first repeatedly failed to recognize the differences between differently located tactile stimuli, which were applied in a consecutive series. Thus he might make the same localization—give the same answer—to each of a long series of tactile stimuli, no consecutive two of which were applied to the same epot. This definitely shows that one of the conditions of the badness of his localization of tactile stimuli was the failure to recognize differently located stimuli as in some manner different; in other words, the element of character in the localization of the tactile stimuli was at fault.

Again, the subject at first repeatedly failed to recognize the similarity between the elements of consecutive series of tactile stimuli which were applied to the same spot. Thus at times he would localize series of similar stimuli upon different spots, no consecutive two of which were the same. Here the element of individuality in the localization of tactile stimuli is at fault; the subject is unable to localize correctly a certain specific stimulus, but his error is not a constant one; when this stimulus is repeated in consecutive series, he localizes it now here, now there.

Thus the disturbance of localization of tactile stimuli in this case was probably a mixed one as regards the different elements in that localization. In the first place there was a definite defect in the elements of character and individuality in the localization of tactile stimuli; and in the second place there was a defect, amounting in some cases to positive error, in the element of position.

Such being the condition of the subject, we set ourselves to train selected spots upon the palmar aspect of one of the fingers. The training was directed to improve the elements of character and individuality of the spots, and to improve the factor of "place on the

finger" in their positions: but no direct attempt was made to improve the factor of "finger" in their positions, nor to improve the elements in the localization of any of the spots on the other fingers.

The result of this training was twofold. In the first place there was a very marked improvement of the localization of tactile stimuli on the trained spots both as compared with the accuracy of that localization on the same finger before the training, and with the accuracy of localization upon the other fingers after the training. In the second place there was a much smaller general improvement of the accuracy of localization on the other fingers as compared with their individual accuracies in the records which were taken before the training of the right index finger was commenced.

Of the three spots which were trained upon this finger, one showed an improvement which was not greater in degree than that of the improvement which occurred in the untrained spots of the other fingers. This was the spot on the basal phalanx—"spot 3." The two remaining spots—those on the distal and middle phalanges—"1" and "2"—showed a very great improvement in the accuracy with which tactile stimuli which were applied to them were localized. If the increase in the proportion of correct answers after the training is expressed as a percentage of the correct answers which were given before the training, we obtained a measure of the degrees to which the different spots improved. The improvement of localization on spot "1" is thus about 62·5 per cent.; that on spot "2" is about 110 per cent.; and that on spot "3" is about 13 per cent.

Thus the three trained spots show very considerable variations in the improvement of localization which they exhibit. We can only say that in the case of two of them that improvement is great, and in the case of the third slight; for although the improvement of spot "1" seems from these percentages to be smaller than that of spot "2," there was no scope for greater improvement; after the training the localizations on spot "1" showed no inaccuracy at all.

The inference from this is that the three trained spots show marked independence amongst themselves, and the improvement of one of them is not accompanied *pari passu* by improvement in the others.

In this experiment the tactile stimuli were applied to similar sets of three spots on the palmar aspect of each finger. Thus the four spots "1" on the four fingers have some resemblance amongst themselves; as have also the four spots "2" and the four spots "3." A slight (apparent) general improvement in the accuracy of the localization of

tactile stimuli upon these spots occurred in the other fingers after the training of the palmar set of spots on the right index finger—was this (apparent) improvement conditioned by that training? Later in this section of the paper we analyse this apparent improvement in the untrained spots. It seems that it was possibly not a true one, but due probably to the disappearance of that type of error in which the subject indicated that he was unable to make a certain localization. Ignoring this correction, we shall here assume that the improvement was a true one.

Had it been conditioned by the training of the spots on the index finger it would have been expected that on the other fingers the improvement occurred chiefly for spots "1" and "2"—those which showed most improvement on the index finger after the training. This was not the case. Of the three spots "1" on the three untrained fingers one (on the fourth digit) showed an improvement of about 14 per cent. only; the two others were actually worse after the training of the index finger. Of the three spots "2" on the untrained fingers, one (on the fifth digit) showed an improvement of about 9·7 per cent. only; the other two were about 12 per cent. and 16 per cent. worse than before. The general improvement in the three untrained fingers was referable to an improvement in the accuracy of the localization of tactile stimuli upon the spots "3"—those which corresponded to the trained spot which showed only a slight improvement.

From this we may infer that the marked improvement in the accuracy of the localization of tactile stimuli upon two of the trained spots was not accompanied by a corresponding improvement of similar spots on the untrained fingers.

This method of analysing the errors of localization and directing the examination, as we here are doing, chiefly to possible interdependencies of errors (or of relative changes in error) is really an investigation of the possible elements in the normal process. If the changes in the accuracy of localization of tactile stimuli upon two different spots show no dependence the one upon the other these two spots to a certain extent must be regarded as functionally independent. It is true that stimuli which are applied to them may have common elements or attributes (such as the common element "right index finger" in the complete localization of two tactile stimuli applied to two different spots upon that finger); but certain of their elements must be peculiar to themselves.

Now, after the training of the spots on the right index finger, our

subject was able to localize stimuli applied to them more accurately than before. It is possible that the effect of the training was to give him a new schema of the cutaneous field upon which these spots lay; or perhaps to give him a schema in which there were only three spots, one far from the palm of the hand, one nearer the palm, and one yet more near. It is clear that such a schema would possibly serve equally well for the placing of tactile stimuli which were applied to another set of three spots on the same finger—a set obtained, as it were, by shifting our three original spots each one half-phalanx length nearer to the palm of the hand. In other words, do nearly similarly located spots on the trained finger take part in the improvement of the accuracy of localization of tactile stimuli which are applied to them?

Our results show that this is not the case. When the accuracy of localization of tactile stimuli is investigated for untrained sets of spots (which are similarly arranged to the trained spots on the finger) no improvement *pari passu* with the improvement in the trained spots occurs in them. Had such an improvement occurred it would have been expected that the "untrained" set of spots on the trained finger would have showed greater accuracy in the localization of tactile stimuli upon them than spots similarly situated on the other fingers: and that, of the three "untrained" spots on the trained finger, that which corresponded to the trained spot "1" would have shown the greatest accuracy of the three. As a matter of fact, we found that the "untrained" spots on the trained finger were far less accurately localized than the corresponding spots on the other fingers, and that, of the three untrained spots on the trained finger, the spot which corresponded to the trained spot "1" was the least accurately localized.

From this we may infer that the improvement in the localization of a set of spots on a finger after training does not extend to an untrained set of spots similarly situated relatively to one another upon the same finger.

In this first part of the experiment we also used a third set of spots on each finger—that set which lay on the dorsal aspect. The three spots of this set corresponded in position exactly (or very nearly exactly) with the ordinary spots on the palmar aspect. The only difference between the two sets was that one was situated upon the palmar and one upon the dorsal aspect of the finger.

Now the investigation of the accuracy of localization in the dorsal sets of spots on the fingers after training of one of the palmar sets of spots presents points of interest. In the first place, the training

of a set of spots on one aspect of a finger is accompanied by an increase of the accuracy with which tactile stimuli are localized upon them. Does this increase apply also to similar spots on the dorsal aspect of the same finger? In the second place, if one effect of the training of the palmar spots on the right index finger is the perfecting of the subject's schema of that part of his cutaneous field, and if at the same time the relation between that part of the field (i.e., palmar surface of right index finger) and neighbouring parts (i.e., palmar surfaces of the other fingers of the same hand) is also improved in the subject, an interesting possibility arises. The subject had before his eyes the palmar aspect of the model's right hand. To this object he related the tactile stimuli which he experienced. The right index finger was improved by education, and that lay to the right of the other three fingers. But when the spots on the back of the hand were tested, the subject looked at the dorsal aspect of the model's right hand, and then the little finger lay to the right of the other three fingers. In other words, when the relative positions in space of the four fingers are alone considered, the little finger occupies the relative position on the dorsal aspect of the pronated hand that the index finger occupies on the palmar aspect of the supinated.

After training the palmar set of spots on the right index finger the accuracy of localization of tactile stimuli on the dorsal sets of spots apparently improved for all the fingers. The (apparent) improvement was greatest for the little finger, about equally great for the index finger and ring finger, and least great for the middle finger; but in no case was the improvement comparable to that which occurred in the case of the trained palmar set of spots itself. In that trained set the chief improvement occurred in the localizing of tactile stimuli upon spots "1" and "2." In the case of the dorsal aspect of the little finger the accuracy of localization of tactile stimuli upon spot "1" was worse than before, but that of spot "2" was better. In the case of the dorsal aspect of the right index finger, improvement occurred for both spots "1" and "2," while the accuracy of localization became worse for spot "3." The improvement of spot "1" here is striking, not for its extent but because no such improvement of this spot occurred on the other fingers (save a small and negligible one on the fourth digit). Of the spots "1" on the palmar aspects of the fingers a great improvement occurred on the trained finger, a comparatively small improvement on the fourth digit again, and the localization of stimuli became worse for this spot on the second and fifth digit.

This would almost indicate that there was in fact an improvement

of localization of tactile stimuli applied to the spots on the dorsal aspect of the trained finger—an improvement conditioned by the training of the palmar set of spots. But there was a very significant difference between the two improvements. In the case of the trained set of spots, the improvement in the accuracy of localizing tactile stimuli applied to one of them was accompanied by a decrease in the number of times stimuli which were applied to the others were thought to be located upon that first spot. In other words, where a larger proportion of the stimuli applied to spot "1" were localized by the subject upon spot "1," a smaller proportion of the stimuli applied to spots "2" and "3" were localized by the subject upon spot "1." When the sets of spots on the untrained fingers were examined after training the right index finger, this did not obtain. For we then found that (with few exceptions) where improvement in the accuracy of localization of tactile stimuli occurred for a certain spot on a certain (untrained) finger, there occurred also an increase in the number of times stimuli applied to the remaining two spots on that finger were wrongly localized upon that certain spot. This relationship of the increase of accuracy in localizing stimuli applied to a spot and the increase in the error of localizing upon it stimuli applied to the other two spots was well marked in the case of the dorsal sets of spots on the right index and little fingers. In other words, whereas in the trained palmar set of spots on the right index finger increase in the accuracy of localizing tactile stimuli applied to spot "1" was accompanied by decrease in the error of localizing upon it stimuli applied to spots "2" and "3": in the untrained dorsal set of spots on that finger increase in the accuracy of localizing tactile stimuli upon spot "1" was accompanied by increase in the error of localizing upon spot "1" stimuli applied to spots "2" and "3."

We are, therefore, most probably justified in assuming that, whereas the great improvement in the localizing tactile stimuli upon the trained spots themselves is referable to the training, the smaller (apparent) improvement for the corresponding untrained spots on the dorsal aspect of the trained finger is not referable to that training, but is conditioned by some more general process. To the consideration of that general improvement we shall return later.

Thus far we have considered various facts which seem to point to the relative independence of the factors which condition the accurate localization of tactile stimuli applied to different spots on the skin, and before passing on we may notice some other phenomena which seem to point to the same conclusion.

There are two kinds of error in localization which may possibly be related to different elements in the process. Of these the first is the tendency to localize upon different spots stimuli which actually are applied consecutively to the same spot on the finger; and the second is the tendency to localize upon one and the same spot stimuli which actually are applied to different spots. The first type of error may be associated with defect in the element of "individuality" in the localization; and the second type may be associated with defect in the element of "character." In investigating these errors various relationships may be examined. For in the first place there may be a relation between the degrees of the two errors in any one finger at any one examination—that is, both errors might constantly be great or both might be small; or the relations between the two might be a reciprocal one, one being small when the other is great—or there may be no constant relation. And, in the second place, if improvement in one or both of these errors is one of the conditions of the improvement of the localization of tactile stimuli applied to the trained set of spots, another relationship may be investigated when the incidences of these errors are examined in the cases of the spots on the untrained fingers—that is, it is possible that a constant relation exists between fall in these types of error in the trained spots and fall in the untrained ones, or that no such relation exists.

There appears to be no constant relationship between the incidences of these two types of error in any one set of spots at any one examination. Sometimes the proportions of the two types of error may both be high; at other times they may both be low; at yet other times one may be low and the other high.

As the one type of error denotes defect in the element of "character" and the other denotes defect in the element of "individuality," this seems to indicate, as far as these present results go, that impairment of the element of "character" in the localization of tactile stimuli which are applied to different spots is not necessarily related, either directly or inversely, with impairment of the element of "individuality."

When we examine the incidences of the different types of error after the training of the palmar set of spots on the right index finger, we found that in the first place there was a marked fall in the error wherein the subject localized two consecutive similarly located stimuli upon two different spots. There was also a fall in the number of series of consecutive similar localizations which the subject made to dissimilarly located stimuli—although the fall was slight if analysed only in

terms of two consecutive similar localizations of stimuli applied to two different spots where the first localization was a correct one. It is probable that the improvement of localization of stimuli applied to the trained set of spots was due to reduction of both these types of error. The first of these two types of error may be termed here "individuality-error," and the second "character-error."

The decline in the incidence of individuality-error for the palmar set of spots on the right index finger was accompanied by an even greater decline for the dorsal (untrained) set of spots on the same finger. This would seem to indicate a relationship between the two sets of spots—so that the training of one of them conditioned an improvement in the localization of tactile stimuli which were applied to the other set. But in the case of the palmar and dorsal sets of spots on the right little finger a great decrease in the incidence of this type of error occurred for one set and an equally great increase in the incidence of this error occurred for the other set. This would hardly be the case if such a relationship between the two sets of spots on a finger existed, and we are perhaps correct in supposing that the training of the palmar set of spots on the right index finger was not the condition of the improvement in this type of error in the case of the dorsal set of spots on the same finger—but no positive conclusion can be drawn.

The individuality-error shows its greatest decline in incidence in the cases of the three sets of spots, the localization of stimuli applied to which showed the greatest improvement (trained palmar spots of right index finger, untrained dorsal sets of spots of index and little fingers), but there is no constant relation between the degree of improvement of localization and degree of fall of this error. The degrees of improvement of localization in the records after training the right index finger were very similar in four of the sets of untrained spots (these degrees may be denoted by the figures 2·3, 3·7, 2·4, and 2·0), but the corresponding degrees of change of the individuality-error showed marked variation (these degrees may be denoted by the figures −1·2, −8·0, +29·8, and +7·9).

After the training of the palmar set of spots on the right index finger there is therefore no constant relationship between the changes in the incidence of the individuality-error; and it may perhaps be inferred that, if the fall in the incidence of that error in the trained set of spots was conditioned by the training, that training conditioned no corresponding fall in the case of the untrained sets of spots.

Our figures for the character-error are not so definite, but in this

case also—as far as the figures go—no constant relationship between improvement in the trained spots and change in the incidence of error in the untrained ones can be drawn.

Another curious absence of relationship occurred in these experiments. After the training of the palmar set of spots on the right index finger the subject greatly improved in his localizations of tactile stimuli which were applied to them when he knew that the stimuli were being applied to these spots and to them alone. It might have been supposed that along with this improvement there would have been an improvement in the accuracy of his localization of stimuli applied to them when the stimuli were not restricted to that finger. That is to say, that when the stimuli were applied to other spots as well as to the trained ones, it would have been expected that at any rate the subject would point to the right index finger each time it was touched. We actually found that in such unrestricted tests, after the training the subject indicated the right index finger less often correctly than before the training. The test of course was not completely unrestricted, it was restricted to twelve spots—three on the palmar aspect of each finger of the right hand. We term it "unrestricted" (meaning "comparatively unrestricted") because it was not restricted to the spots on a single finger.

The inference from this is, that in the improvement of the localization of tactile stimuli applied to the trained set of spots on the right index finger, improvement of the element of "finger" in the "position" was not one of the factors.

So far, therefore, it would seem that the improvement in the localization of tactile stimuli applied to the trained spots is not only restricted to that set of spots, but that it is a special and independent process for each of the trained spots. We must now refer to the results of one test which seem to contradict this.

When the subject localized stimuli applied to the ordinary spots on the palmar surfaces of the four right fingers without knowing to which spot or to which finger the stimulus was applied, it was found that the accuracy of localizing the spots (without taking account of the accuracy of localizing the finger on which they lay) was much greater than it was expected it would be. We had thought it possible that the proportion of correct replies to this part of the questions put to the subject would approximate to the average of the proportions for the spots on the four fingers when these were tested separately in the ordinary test—where the subject knew to which finger the stimuli were being applied. We thus expected that, where the localization for the

spots of one finger was very inaccurate, and where it was comparatively accurate for the spots of another finger, as these spots were equally often touched in the unrestricted finger test the accuracy of localization there would be the mean of the accuracies for the separate fingers in restricted finger test. Indeed it might have been expected to be less if hazard came into the localization; for the hazard error in the test of a single finger is 66·7 per cent., whereas in the unrestricted finger test it is 91·7 per cent. As a matter of fact, we found that this error was much less than the average of the errors for all the fingers in the restricted test, and that actually it approximated, both before and after training, most closely to the error of the most accurately localized set of spots in the restricted test.

This result (which in this case was most striking, but which we have not obtained in another similar case) was quite an unexpected one. After the training of the palmar set of spots on the right index finger that was the most accurately localized set, and the average error of localization fell most markedly for it. In the unrestricted test there was a corresponding increase in the accuracy of localization of the spots touched as regards the place on the finger (that is, as regards the position of the spot in the set) and irrespective of the accuracy of localizing the finger.

The inference from this would seem to be that as regards this specific case, the training of one set of spots was accompanied by an improvement of the element "place on finger" in the localization of tactile stimuli which were applied to them and that this improvement extended to other and untrained spots, but only (or only markedly) when the test was not restricted to a single finger.

It is not difficult to see how such a general improvement may have occurred. The three spots on each finger occupied similar positions relatively to the parts of the finger—one spot lay in the middle of each obvious part or segment of the finger. The training of the spots of one set no doubt improved the subject's schema of their positions; it improved, as it were, the element "place on finger" in the "positions" of the touches. But that element is common to the spots of the untrained sets, and its improvement for one set may well have conditioned improvement for other and untrained sets of spots. The difficulty is to see how, in this case and at this period in the experiment, the improvement occurred only when the test was one unrestricted as regards the fingers to which the tactile stimuli were applied—but restricted to the three spots on each finger.

The occurrence of "positive error" in the localization of the finger (apart from the localization of the place of the spot on the finger) in this test gives us a hint as to the reason for this curious restriction of the improvement. In the unrestricted test it was found that the subject more often localized the tactile stimuli upon certain fingers than upon others, and that the error in localizing finger alone was in some cases considerably greater than the error of hazard. In other words the subject had a definite bias against the localization of stimuli upon certain fingers. The restriction of the test to a single finger (when that was done) no doubt brought in a complicating factor; for the subject knew that the tactile stimuli were applied to a certain finger, and might yet feel that the touches were on another finger. In making his decision, for instance, of the localization of a touch applied to spot "2" on a certain finger, he might think that the touch (which felt, for example, as if it was on spot "2" of another finger) most resembled one applied to spot "3" on the finger to which he knew that the stimuli were being applied. Thus he might make an incorrect localization of the place of the touch upon the finger, although—had the test been unrestricted as regards the fingers—he would have placed the touch in the correct position upon the finger, but on the wrong finger. It is therefore possible that in the unrestricted finger test a complication was removed, and this common element of "place on finger" in the "position" of the touches was so freed that the improvement in it conditioned by the training of one set of spots was extended to others.

We may now consider the nature of the improvement of localization of tactile stimuli which followed the training of the spots on the palmar aspect of the right index finger. That improvement occurred not only in the case of the trained set of spots, but also in the case of some of the sets of spots on the untrained fingers.

In the case of the trained set of spots the records show a decrease both in the character-error and in the individuality-error after the training. Thus, after that training, when tactile stimuli were applied to the three spots in such an order that no two consecutive stimuli were applied to the same spot, on the whole there were fewer consecutive similar answers given—on fewer occasions after the training were consecutive dissimilar stimuli localized by the subject upon the same spot on the model. At the same time, when tactile stimuli were applied to the three spots in such a manner that two or more consecutive stimuli were given to the same spot, there were, upon the whole, fewer consecutive dissimilar answers.

But in our results there was also a reduction in one of the position-errors. The abolition of the character-error—so that the subject was always able to say correctly whether or not a certain tactile stimulus was applied to that same spot on the skin to which an immediately preceding stimulus had just been applied—if unaccompanied by improvement in the other factors in localization would easily be recognized in the records. For where series of tactile stimuli were given so that no consecutive two were applied to the same spot, the answers would also show series in which no consecutive two answers were similar, but tactile stimuli applied at different times to any one spot would be localized now upon one spot of the three, and now upon another. The error in the localization of tactile stimuli applied to a certain spot of the three would be the error of hazard—66·7 per cent., and of the answers to these stimuli 33·3 per cent. would be localizations on each of the two possible wrong positions. If, however, there was also abolition of the individuality-error the errors of localization would be expected to become constant. Stimuli applied to spot "3" might thus always be localized upon spot "2," and so on. In such a case the error of localization of stimuli applied to a certain spot in a series of records would be the error of hazard if the individuality of the tactile stimuli applied to that spot varied from day to day. In any case the answer to stimuli applied to a spot would be expected to be constant for any one record, and the average error for all the spots tested would be expected to be the error of hazard.

Our records give no approximation in error to the error of hazard after the training of the spots. The inference is that along with the reduction of the character-error and the individuality-error there is a reduction of the position-error. As reduction of the character-error occurred to an even greater extent in the case of certain of the untrained sets of spots; and as reduction of the individuality-error occurred to an even greater extent in the case of certain other sets of untrained spots; but, as, nevertheless, the reduction of the total error of localization of the untrained sets of spots was in no case comparable with the reduction of this error in the case of the trained set of spots, we are inclined to think that the improvement in the localization of tactile stimuli applied to the trained spots was chiefly due to improvement in the element of "position."

Improvement in the localization of stimuli which are applied to a certain spot would be expected to have a reverse as well as an obverse side. In the first place, stimuli applied to that spot are less often

localized by the subject upon other spots: and in the second place, stimuli applied to other spots would be expected to be less often localized by the subject upon the spot. If the character and the individuality of stimuli applied to the spots are alike improved, and if at the same time there is an improvement in the position—in the correct placing upon the finger—of each of these stimuli, the stimuli applied to a certain spot will less often be localized upon another, and the stimuli applied to the other spots less often upon the first.

Of the three trained spots, spot "1" showed a very definite improvement in the localization of tactile stimuli which were applied to it. They were always correctly localized after the training– so that the error of localizing upon the other two spots touches which were applied to spot "1" fell to zero. At the same time there was a reduction of the errors of localizing stimuli applied to either of the other two spots upon this spot. After the training spot "2" showed also a great improvement in the localization of tactile stimuli applied to it. The error of localizing touches of this spot upon either of the other two spots ("1" and "3") fell considerably; but, while there was a reduction of the error of localizing touches of spot "1" upon this spot (a reduction to zero), there was actually a small increase of the error of localizing touches of spot "3" upon it. Spot "3" itself showed a comparatively small improvement in the localization of tactile stimuli applied to it. The error of localizing touches of this spot upon spot "1" fell slightly (the reduction being much less than that of the error of localizing touches of spot "2" upon spot "1"), but the error of localizing touches of spot "3" upon spot "2" actually rose slightly. The errors of localizing touches of spot "1" and "2" upon this spot "3" showed, as we have said above, great reductions.

From these differences in the improvement of the accuracy of localization of tactile stimuli upon spots "1" and "2," on the one hand, and spot "3" upon the other, it may perhaps be inferred that the training comparatively failed in the case of the last, and that the slight improvement was due to some other process than that which occurred in the two former. Again it must seem as if the obverse and the reverse of the improvement in the localization of tactile stimuli which are applied to a trained spot are not interdependent. Thus a great improvement in the localization of tactile stimuli which are applied to a certain spot (spot "2") is accompanied by a decrease in the error of localizing these stimuli upon a certain other spot (spot "3"), but there is no corresponding decrease (and indeed there is actually an increase) in the error of

localizing tactile stimuli which are applied to that certain other spot ("3") upon the first spot ("2"). The subject as it were recognizes more clearly touches which are applied to a certain spot, but this more clear recognition does not necessarily help him to realize that touches which are applied to another spot are not applied to the first spot. In other words the improvement of localization of tactile stimuli which are applied to a certain spot is not necessarily accompanied by a corresponding improvement for other spots on the same aspect of the same finger. It would appear as if each spot is improved for itself without reference to the others. Where the improvement occurs in two spots there is a mutual relationship—"obverse" and "reverse"—in the reduction of the errors of localization of the two; but where only one of a pair of spots shows a great improvement no such mutual relationship may occur.

In addition to the special improvement in the localization of tactile stimuli which occurred in the case of the trained spots, there occurred a less marked general (apparent) improvement in localization for the untrained sets of spots on the other fingers—and on the dorsal aspects of all the fingers. In what did this consist?

A most marked change which synchronized with the period of training was the disappearance of error due to "confusion." Before the training the subject repeatedly indicated that he was unable to come to a decision with regard to the localization of certain stimuli. After the training, on scarce an occasion did he fail to make a localization of some sort, and the disappearance of this source of error was a general one—it extended to stimuli applied to the untrained as well as to the trained sets of spots. It is clear that even if the subject had hazarded his localizations of the touches of which he was not certain, this would have led to a reduction of the percentage error of localization—for a certain proportion of these touches would then have been bound to be correct. If, however, we speculate that the "confused" touches were (after the disappearance of the "confusion" error from the records) really localized in the same proportion (as between "right" and "wrong") as the definitely localized touches before the disappearance of the "confusion" error; and, having found the proportion of the "confused" answers which corresponds with the proportion of wrong definite localizations before the disappearance of "confusion," we then add the two together, it is found that the resultant corresponds very definitely with the proportion of definite wrong localizations made in the records after the disappearance of the "confusion" error as far as the untrained sets of spots are concerned. This is demonstrated in Table

XXXIII. It is there seen that except in the case of the trained set of spots on the palmar aspect of the right index finger the percentage error of localization for the untrained sets of spots (after the disappearance of the "confusion" error) is very nearly what it would have been had the same proportion of the previously "confused" touches, as of the previously definitely localized touches, been wrongly localized. In the localization of touches on the palmar sets of spots the actual error is slightly greater than it would have been if this had been exactly the case; a yet closer approximation occurs in the case of the dorsal sets of spots, but only in the case of the dorsal set of spots on the little finger ("R 5") is the actual error less than the computed.

It will be remembered that there was some evidence to show that in the case of one of the trained spots the improvement of localization was probably not very largely conditioned by the training—spot "3." In Table XXXIII the figures for the three individual spots of the trained set are also given. It is seen that in the case of spots "1" and "2" the actual error of localization after the training (and after the disappearance of the "confusion" error) is very much less than it would have been if the decline in error had been due solely to the correct localization of a proportion of the touches which before had been "confused." But in the case of spot "3" we again obtain a close approximation between the actual error and the error computed as above (that is, by adding to the proportion of definitely wrongly localized touches before the training the corresponding proportion of the un-localized or "confused" touches). Here, however, the actual error is slightly less than the computed.

This all seems to point to the fact that the apparent improvement in the localization of tactile stimuli upon the untrained spots (and upon one of the trained ones) is really due to the disappearance of the "confusion" error—that it is not a real general improvement. The figures throw into an extremely strong light the great change which took place in the case of two of the trained spots—a change which we think can be ascribed to the training. We think that the apparent improvement in the cases of the untrained sets of spots is, at this stage in the experiment, not a real one; but we say this with the knowledge that later a real improvement occurred.

This seems to dispose of the question of the apparent general improvement in localization of touches applied to the untrained sets of spots, but certain changes took place in the incidence of error amongst them after the training of the right index finger—and these changes are of interest. In some cases the proportions of correct localizations for

the touches applied to the individual spots on the untrained fingers showed differences in the records before and after the training of the palmar set of spots on the right index finger. But, whereas in the case of the trained spots the increase in the proportion of correct answers for a spot was, on the whole, accompanied by decrease in the wrong localizations upon it of touches applied to other spots, in the case of the untrained spots a rise in the proportion of correct localizations for a certain spot was almost always accompanied by a rise in the proportion of times touches applied to other spots were localized upon it. This really means that, after the training, the subject was more apt to place any tactile stimulus upon a certain spot than before it. Thus in the cases of the dorsal sets of spots there was a greater tendency to give the answer spot "2" (whether it was right or wrong) after the training than before it. The increase in the proportion of times spot "2" was given as an answer (which was accompanied by an increase in the proportion of correct localizations of touches applied to that spot) was accompanied by a decrease in the proportion of answers spot "1," and by a decrease in the proportion of answers spot "3." It occurred, as it were, at the expense of the answers spot "1" and spot "3," and at the expense of the answer "confused." Here we have a definite tendency to a certain localization—which would not be expected to be the case had the answers merely been selected by hazard.

In connection with this definite change in the numerical proportion of the different answers to the touches it must, however, be noticed that both before and after the training of the right index finger there was a greater tendency to make the wrong localizations of touches of spot "1" upon spot "2" than upon spot "3"; and a corresponding greater tendency to make the wrong localizations of touches of spot "3" upon spot "2" than upon spot "1." That is to say, that when one of the terminal spots of the set of three was wrongly localized it was more often referred to the middle spot—that nearest to it—than to the spot at the other end of the set. Had a general improvement occurred in the localization of touches upon the untrained spots it might have been expected that, along with a greater proportion of correct answers to touches upon each of the terminal spots of the sets, there would have been a reduction of the incorrect references to the spot at the other end of the set and an increase in the incorrect references to the middle spot—as it were, a partial improvement in the accuracy of localization. Had this occurred the number of answers spot "2" both to touches

applied to that spot itself and to touches applied to the spots at the two ends of the set (spots "1 and 3") would have increased. But as a matter of fact the increase in the number of answers spot "2" in the case of the untrained dorsal sets of spots increased along with a decrease in the correct localizations of touches applied either to spot "1" or spot "3" or both to the touches applied to these two spots. We are, therefore, justified in the assumption that the increase in the number of answers spot "2" in the case of tactile stimuli applied to these untrained sets of spots was not due to a partial general improvement of localization, but to a definite tendency to refer the touches rather to that spot than to the others—an approximation to "positive error."

On the whole, then, we may say that proper analysis seems to eliminate the occurrence of a general improvement of the localization of tactile stimuli which were applied to the untrained sets of spots (and perhaps also to one of the trained spots): and that it seems to show that the improvement in the case of two of the trained spots was due either to the effect of the training or to some other change which did not affect the untrained sets of spots.

It is thus possible that the conditions of the experiment more favoured the accuracy of localization of touches upon the set of spots which we selected for training than that of the touches upon the untrained sets of spots; and it is also possible that the conditions of the cortical (or cerebral) lesion were not stable, and that by chance the change effected an increase of the accuracy of localization for the trained spots only.

Thus, in the first place, we investigated the fingers in a constant order. First, the palmar surfaces of two of the normal fingers were tested; then, in order, the palmar surfaces of the four affected fingers—beginning with the right index finger; then the dorsal surfaces of two of the normal fingers; and finally the dorsal surfaces of the four affected fingers—again commencing with the right index finger.

It is thus possible that a process of "general fatigue" may have so complicated the results that the fingers first tested may have appeared more accurate in localization than those last tested. But our records give little indication that this was the case. In the first place the records before the training of the right index finger should then have shown the same progressive changes as those taken after it. This is not the case. The first affected finger to be tested shows then greater inaccuracy than the second: the third than the fourth. In the second place the different fingers might have been expected to show a definite

descending order of improvement of localization in the order in which they were tested. This, again, is not the case. Thirdly, in this part of the experiment we constantly used as a control one of the normal fingers, testing the accuracy of localization of tactile stimuli upon it at the end of the record, when general fatigue would be expected to be at its maximum. The accuracy was seldom found to be less than at the commencement of the test. Fourthly, on one occasion we repeated the test of the right index finger at the end of the record, and the accuracy of localization was then found to be greater than before. On the whole, we may say that, although there probably was some process of general fatigue, it cannot be regarded as the explanation of our results.

But we cannot, on the evidence furnished by this part of the experiment alone, eliminate the possibility that the improvement of the localization of tactile stimuli applied to the trained spots may have been due to some change in the condition of the central lesion—a change associated only by chance with the training of the spots. We think, however, that this possibility must be eliminated in view of the data we obtained in subsequent parts of the experiment. For, later on in it we selected another finger—the fourth right digit—and found that improvement in the localization of tactile stimuli applied to its palmar set of spots also occurred during and after training. As the error of localization of tactile stimuli upon the spots of this finger was large before that training—as it was, indeed, larger than that of any other set of spots in this first part of the experiment—and, as after the training, it became only less small than the error in the case of the finger which we first trained, we think that this disposes of the possibility that the results which we have described here were due to a change not connected with the training. We do not claim, however, that no such change, or that no general improvement in the localization of tactile stimuli occurred throughout the whole course of the experiment.

V.—Analysis of the First Series of Tests, during which an Attempt to educate Spots in Accuracy of Localization of Tactile Stimuli was made.

(1) *In General.*

In this series of tests two sets of three different spots were selected for examination in each of the second, third, fourth, and fifth digits of the affected right hand, and on the third and fourth digits of the

"normal" left hand. Of these two sets, in each case one lay on the palmar aspect and one on the dorsal; the three spots of each set lay on the skin of the finger in its dorsi-ventral axial plane, one being opposite the mid-point of each of the three phalanges. For the purposes of a small number of tests, a third set of three spots was chosen on the palmar aspects of each of the four fingers of the right hand. These also lay on the skin in the dorsi-ventral axial plane of the finger, but they were placed on the folds of skin at which the finger bends, and which lie near to the metacarpal-phalangeal and the two interphalangeal joints.

In the records care had not been taken to assure that each spot on a finger in the test was stimulated the same number of times, and the number of times a specific spot was stimulated in different records varied. As the experiment proceeded, we found that the accuracy of localization of tactile stimuli was not the same for the three different spots on a finger—it was, for instance, sometimes most defective for spot "3" (that nearest the trunk) and least defective for spot "1" (that nearest the tip of the finger). It is thus clear that a record in which spot "1" is stimulated more often than spots "2" or "3" may give a different percentage of error than one in which spot "3" is most often stimulated. In estimating the error of localization in a record in this series of observations, we have, therefore, used the "average percentage error" which is obtained by finding the "percentage error" for each of the three individual spots, and taking the average of the three percentages. As there was not, however, a large preponderance of the times any one individual spot was stimulated in any one record, this does not markedly differ from the percentage error of the record; the difference between the percentage error and the "average percentage error" in the records is usually less than 1 per cent., and rarely greater than 2 per cent.

(2) *On the Records which were taken before Training commenced.*

For the first to the fifth day of the series the tests were applied in the ordinary manner to the spots on the palmar and dorsal aspects of the fingers. The following table, in which L 3 and L 4 stand for the digits of the left hand, and R 2, R 3, &c., for those of the affected right hand, gives the "average percentage error" of localization for the spots on the palmar and dorsal aspects of the different fingers on the five different days—twenty-five stimuli applied in an indiscriminate order being given to the three spots on each aspect of each of the fingers which were tested:—

TABLE I.—"Average Error per cent." for Localization of Tactile Stimuli on the Palmar and Dorsal Sets of Spots of each Finger on each of the Five Days which immediately preceded the attempt to Train the Palmar Spots on the Right Index Finger. Each Figure is Calculated from Twenty-five Answers—except the Averages and Mean Variations in the Right-hand Columns.

Finger	Day of Experiment 1	2	3	4	5	Average	Mean variation
Palmar L 3	7·4	0·0	0·0	0·0	0·0	= 1·48	2·37
L 4	0·0	0·0	14·3	0·0	8·3	= 4·52	5·42
R 2	35·4	41·8	50·9	44·0	41·4	= 42·70	3·80
R 3	31·7	17·6	38·7	25·9	46·0	= 31·98	8·30
R 4	39·0	40·0	46·3	47·3	61·1	= 46·72	5·96
R 5	46·0	44·0	33·3	36·9	55·1	= 43·05	6·37
Dorsal L 3	0·0	0·0	0·0	0·0	3·3	= 0·66	1·06
L 4	4·3	3·3	0·0	2·6	0·0	= 2·04	1·63
R 2	25·4	30·7	20·4	44·4	21·9	= 28·56	7·19
R 3	19·0	11·4	20·9	21·7	20·8	= 18·76	2·94
R 4	43·5	15·0	17·6	26·9	31·0	= 26·80	8·40
R 5	27·0	27·8	20·0	53·1	35·2	= 32·62	9·22

It will be observed that for their palmar aspects the arrangement of the right digits in order of decreasing accuracy of localization of tactile stimuli is: R 3, R 2, R 5, R 4; the corresponding order for the dorsal aspects is: R 3, R 4, R 2, R 5; and the most accurate finger for either aspect is R 3.

The second table gives the average percentage error for each individual spot on each finger in this same series of records:—

TABLE II.—The Total Percentage Error of Localization for each of Spots "1," "2," and "3" on the Palmar and Dorsal Aspects of each of the Fingers examined on the Five Days which immediately preceded the Training. Each Percentage is Calculated from about Forty-two Observations.

Finger	Number of Individual Spot. 1	2	3
Palmar L 3	0·00	2·17	2·63
L 4	0·00	0·00	15·38
R 2	32·43	62·50	35·00
R 3	13·33	26·32	59·52
R 4	25·71	31·37	79·49
R 5	37·78	46·67	40·00
Dorsal L 3	2·38	0·00	0·00
L 4	4·65	1·82	0·00
R 2	45·95	25·00	15·00
R 3	15·56	34·21	9·52
R 4	31·43	33·33	20·51
R 5	37·78	40·00	17·14

It will be observed that in the case of fingers R 3, R 4, the accuracy of the localization of tactile stimuli on spot "3" (that nearest the

trunk) on the palmar aspect is markedly less than that on spots "1" and "2"; while the accuracy of the localization on spot "1" is markedly greater than that on spot "2." In the case of the other two fingers (palmar aspects) the accuracy of localization of tactile stimuli is worst for spot "2," and about equal on spots "1" and "3." When the figures for the dorsal aspects of the right digits are examined, it is seen that in every case the accuracy of localization is least defective on spot "3" (that nearest the trunk), and, except in the case of R 2, most defective on spot "2."

The average percentage error for the localization of tactile stimuli on spot "3" of the palmar aspect of the fourth right digit—R 4—is a figure of interest. That percentage is 79·49—markedly greater than the error of hazard, which is here 66·67 per cent. The following table expands this figure :—

TABLE III.—Percentage Error of Localization for Spot "3" on the Palmar Aspect of the Fourth Right Digit, "A" in each of the Five Records before, and "B" in each of the Five Records after, "Training" the Second Right Digit.

Record	A	Record	B
1	62·50	9	100·00
2	75·00	10	50·00
3	100·00	11	54·55
4	90·91	12	66·67
5	75·00	13	77·78

It will be seen that on only one occasion—the first—was the average error for this spot less than that of hazard. This is an instance of "positive" error in localization. It is markedly greater than the error which would occur if the subject had no idea which of the three spots was touched, and indicated any one of them indiscriminately by chance.

The kind of answer which was given in these cases to a tactile stimulus which was applied to spot "3" is of interest. Each time the spot was touched the subject gave one of four possible answers with regard to its localization. In the first place the answer might be correct, and the subject then indicated correctly the corresponding spot on the hand of the model. In the second place the subject might be unable to make a localization (he then tapped the table). While in the third and fourth places the subject might make a wrong localization—indicating either spot "1" or spot "2" on the model as that corresponding to the spot touched. The following table gives the average number of times per cent. each of these answers was given :—

TABLE IV.—Analysis of the kind of Answers given when Spot "3" on the Palmar Aspect of the Right Fourth Digit was touched in the Records detailed in Table IV, giving Total Numbers and Percentages for the Two Sets of Records, "A" and "B."

Answer	A (Records 1 to 5) No.	A Per cent.	B (Records 9 to 13) No.	B Per cent.
Right—i.e., "3"	8	20·51	14	30·43
Confused	4	10·25	0	0·00
"1" instead of "3"	1	2·56	2	4·35
"2" instead of "3"	26	66·67	30	65·22
	39	99·99	46	100·00

Had they been selected by hazard the percentage in each case would have been about 25·00; as it is, it is seen that the wrong answer "2" for "3" is given in two-thirds of the cases. The positive error in the localization of tactile stimuli applied to spot "3" on the subject is, therefore, very largely conditioned by his mistaking the locus of the stimulus for that of stimuli applied to spot "2."

The number of times "confusion" occurred in the record for the palmar and for the dorsal set of spots of each finger on each of the first five days is given in the table below. The total number of stimulations in each case was 25 for the set of palmar spots, and the same number for the dorsal:—

TABLE V.—The number of times (out of Twenty-five Observations in each case) no Localization could be made—"Confusion"—for each of the Palmar and Dorsal Sets of Spots of each Finger on each of the Five Days which immediately preceded the Training.

		Day of Experiment 1	2	3	4	5
Palmar	L 3	0	0	0	0	0
	L 4	0	0	0	0	0
	R 2	0	2	3	6	8
	R 3	0	0	2	0	5
	R 4	0	1	3	4	5
	R 5	0	0	3	3	4
Dorsal	L 3	0	0	0	0	0
	L 4	0	0	0	0	0
	R 2	0	1	2	8	3
	R 3	0	1	2	2	6
	R 4	0	2	1	3	5
	R 5	0	1	2	7	6

The apparent absence of "confusion" on the first day is due to the fact that the subject did not clearly realize that he was to tap the table when he was unable to make a localization; on this day it is probable that a certain number of his answers were guesses. We think that

this is the only occasion in the experiments on which he guessed an answer.

A remarkable feature of the early experiments was that on the one hand the subject repeatedly localized successive differently located tactile stimuli on the same spot, and that, on the other hand, he repeatedly localized successive similarly located tactile stimuli on different spots.

It is difficult to make an analysis of these types of error. It may be attempted by scrutinizing the answers to consecutive similar pairs of tactile stimuli, but this makes no account of instances in which different replies are given in series of similar stimuli; or in which the same reply is made many times in succession when different spots are touched. We have observed a marked tendency for a series of similar replies to be given to a series of different tactile stimuli; and when a series of similar stimuli has been given there is a tendency for the type of reply to persist, and for the subject thus wrongly to localize a dissimilar stimulus which is applied immediately after the series.

In the following table the number of times two different answers were given to two consecutive similar stimuli is given as a percentage of the number of times pairs of similar stimuli were given in a test—these percentages being given for the dorsal and palmar sets of spots on each finger on the first five days of the experiment:—

TABLE VI.—THE PERCENTAGE NUMBER OF TIMES PAIRS OF TWO CONSECUTIVELY SIMILARLY LOCATED STIMULI WERE LOCALIZED ON TWO DIFFERENT SPOTS; GIVEN FOR EACH ASPECT OF EACH FINGER ON EACH OF THE FIVE DAYS WHICH IMMEDIATELY PRECEDED THE TRAINING.

		Day of Experiment.													
	Finger	1		2		3		4		5		Average 8·0		Mean variation	
Palmar	L 3	40·0	..	0·0	..	0·0	..	0·0	..	0·0	=	8·0	..	12·8	
	L 4	0·0	..	0·0	..	25·0	..	0·0	..	0·0	=	5·0	..	8·0	
	R 2	50·0	..	20·0	..	20·0	..	60·0	..	50·0	=	40·0	..	16·0	
	R 3	40·0	..	0·0	..	50·0	..	20·0	..	62·5	=	34·5	..	19·6	
	R 4	33·3	..	50·0	..	25·0	..	33·3	..	20·0	=	32·3	..	7·9	
	R 5	16·7	..	11·1	..	28·6	..	25·0	..	60·0	=	28·3	..	12·8	
Dorsal	L 3	0·0	..	0·0	..	0·0	..	0·0	..	14·3	=	2·9	..	4·6	
	L 4	12·5	..	0·0	..	0·0	..	25·0	..	0·0	=	7·5	..	9·0	
	R 2	25·0	..	60·0	..	20·0	..	60·0	..	25·0	=	38·0	..	17·6	
	R 3	0·0	..	25·0	..	16·7	..	40·0	..	37·5	=	23·8	..	12·4	
	R 4	33·3	..	0·0	..	37·5	..	16·7	..	40·0	=	25·5	..	13·7	
	R 5	0·0	..	44·4	..	57·1	..	100·0	..	40·0	=	48·3	..	24·2	

In Table VII the number of times two consecutive stimuli applied to different spots were localized by the subject upon the same spot is given for each record on each of the first five days of the experiment. In

each record of 25 successive tactile stimuli there were about twenty occasions in which two successive stimuli were applied to different spots on the subject :—

TABLE VII.—The number of times Two Different Consecutive Stimuli were Localized upon the Same Spot—given for the Palmar and Dorsal Sets of Spots of each Finger on each of the Five Days which immediately preceded the Training.

Finger	Day of Experiment 1	2	3	4	5	Average	Mean variation
Palmar L 3	0	0	0	0	0	0·0	0·0
L 4	0	0	2	0	2	0·8	0·96
R 2	8	9	5	1	4	5·4	2·5
R 3	6	5	3	7	3	4·8	1·4
R 4	6	10	2	4	6	5·6	2·1
R 5	8	4	5	8	9	6·8	1·8
Dorsal L 3	0	0	0	0	0	0·0	0·0
L 4	1	2	0	1	0	0·8	0·6
R 2	5	4	3	4	3	3·8	0·6
R 3	5	2	6	4	0	3·4	1·9
R 4	8	3	4	3	3	4·2	1·5
R 5	6	5	4	5	2	4·4	1·1

The variation of the results here is in part due to the variation in the number of times in a record the subject was unable to make a localization—as these have not been included in the table.

But, with regard to this type of error, it must be noted that it is of two distinct forms, one of which may be regarded as a more inaccurate type than the other. Thus when two consecutive tactile stimuli are applied to two specific different spots on the skin the subject may localize the first wrongly, but may "correct" his error at the second stimulus (if that happens to be applied to the spot to which the subject had localized the first stimulus). Thus similar replies may be given to two different stimuli. But the error would be one of a different type if the subject correctly localized the first stimulus and then incorrectly localized the second stimulus (applied to another spot) on the same spot as the first. But here also his replies to differently located stimuli would be similar. In other words, when the instances in which the subject replies with the same answer to two consecutive differently located stimuli are examined a distinction should be drawn between those cases in which the first of the two stimuli is correctly localized and those in which the second is. In the following table (VIII) the numbers of occasions on which the subject correctly localized the first of a pair of different stimuli and then located the second stimulus at the same spot as the first are given:—

TABLE VIII.—The number of times the Second of a Pair of Dissimilar Consecutive Stimuli was Wrongly Localized on the Same Spot as that on which the First of the Pair had been Correctly Localized—given for the Palmar and Dorsal Sets of Spots of each Finger on each of the Five Days which immediately preceded the Training. The numbers are out of about Twenty Observations each.

| | | Day of Experiment | | |

On the third of these days we gave the training in the morning, and thereafter repeated the test of the accuracy of localization of tactile stimuli on the trained spots after each interval of one hour which elapsed. The percentage error of localization gradually decreased (having been considerably higher immediately after the training) and at its least was about one-third only of the lowest previous error on this finger. Table IX gives these average percentage errors.

But when the spots on the palmar aspects of the other fingers were tested at the end of the day, it was found that improvement of the accuracy of localization of tactile stimuli had occurred in every case. This is shown in Table X:—

TABLE X.—"AVERAGE ERROR PER CENT." OF LOCALIZATION OF THE SPOTS ON THE PALMAR ASPECTS OF EACH OF THE FOUR RIGHT FINGERS BEFORE AND AFTER THE RECORDS GIVEN IN TABLE IX.

	Finger		Before		After
Palmar	L 3	..	0·0	..	3·0
	R 2	..	37·2	..	25·0
	R 3	..	42·9	..	30·1
	R 4	..	50·0	..	23·3
	R 5	..	57·2	..	41·1

TABLE XI.—"AVERAGE ERROR PER CENT." OF LOCALIZATION OF THE SPOTS ON THE PALMAR ASPECT OF THE RIGHT INDEX FINGER AT DIFFERENT INTERVALS OF TIME THROUGHOUT THE DAY.

Time		Four-hour (9)		Three-hour (10)		Half-hour (12)
10 a.m.	..		..	15·1	..	7·6
10.30 ,,	..	16·7	..	—	..	—
11 ,,	..	—	..	—	..	—
11.30 ,,	..	—	..	—	..	23·1
12 m.d.	..	—	..	—	..	27·9
		Dinner.				
12.30 p.m.	..		..	—	..	26·1 (Finished dinner)
1 ,,	..		..	7·2	..	28·1 (Half pipe)
1.30 ,,	..	—	..	—	..	11·4
2 ,,	..		..	—	..	25·8 (Pipe)
2.30 ,,	..	4·8	..		..	27·2
3 ,,	..	—	..	—	..	25·0
3.30 ,,	..	—	..	—	..	16·9 (Pipe)
4 ,,	..	—	..	7·9	..	28·6 (Sleeping)
4.30 ,,	..	—	..	—	..	15·9
5 ,,	..	—	..		..	36·7
		Tea.				
5.30 ,,	..	—	..	—	..	13·3
6 ,,	..	—	..	—	..	34·5 (Pipe)
6.30 ,,	..	13·4	..	—	..	26·4
7 ,,	.	—	..	12·5	..	34·3

After this training was completed we tested, on different days, the accuracy of the localization of tactile stimuli for the "trained" spots on the palmar aspect of the right index finger at different intervals of time throughout the day. Table XI gives the results obtained on three occasions—the intervals between tests being four hours, three hours, and half hours respectively. It will be seen that there is no marked progressive change in accuracy during the day—except that the answers appear generally to be less accurate in the late evening :—

On the occasion of the half-hourly test the accuracy of localization was tested for the corresponding spots on all four right fingers before and after the experiment. The accuracy seems to be markedly less for the second and third digits after it (Table XII). The accuracy of localization was tested at the same time for the set of "untrained" spots on the palmar aspects of the same fingers (those spots which lay on the folds of skin near the joints). Comparatively little change is seen to occur in the case of the second and third digits : but there is a curious reversal of degree of accuracy in the case of the fourth and fifth :—

TABLE XII.—"Average Error per cent." of Localization of the Spots on the Palmar Aspects of all the Four Right Digits before and after the Records given in the Third Column of Table II.

Finger		Before		After
Palmar L 3	..	13·9	..	0·0
R 2	..	7·6	..	34·3
R 3	..	18·8	..	29·2
R 4	..	43·0	..	47·7
R 5	..	43·9	..	31·7

"Average Error per cent." for the "Untrained" Spots on the Palmar Aspects of the same Fingers before and after these Records.

Finger		Before		After
Palmar L 3	..	18·0	..	4·8
R 2	..	60·0	..	65·3
R 3	..	37·6	..	35·2
R 4	..	28·7	..	55·6
R 5	..	50·8	..	7·6

On the three mornings of the days which immediately followed each of these three trainings the accuracy of localization was tested for the dorsal and palmar sets of spots of all the fingers used in the experiments (Table XIII).

There was a very slight progressive increase in the accuracy of localization of the trained spots of the right index finger ; but no regular change in the case of the spots on the back of it, nor in the case of the palmar or dorsal sets of spots on any of the other fingers.

TABLE XIII.—"Average Error per cent." of Localization of the Spots on the Palmar and Dorsal Aspects of each Finger on the Three Days, each of which immediately succeeded a "Training" of the Palmar Set of Spots on the Right Index Finger. Each Figure calculated from Twenty-five [illegible]rvations.

		Day of Experiment.		
Finger		6	7	8
Palmar	L 3	8·5	0·0	4·2
	L 4	0·0	0·0	0·0
	R 2	39·7	37·2	32·6
	R 3	46·3	42·9	46·6
	R 4	42·0	50·0	37·0
	R 5	21·1	57·2	27·8
Dorsal	L 3	3·7	0·0	0·0
	L 4	0·0	0·0	0·0
	R 2	28·0	13·1	37·6
	R 3	31·2	3·7	35·4
	R 4	32·9	26·7	32·8
	R 5	18·8	25·6	39·3

(4) *On the Records which were taken during the Five Days which immediately succeeded the first Period of Training.*

The ordinary tests were continued on the five days which immediately followed the first attempted training in this experiment. The general results obtained on these days are given in the following table (XIV), where they are expressed as "average error per cent." :—

TABLE XIV.—"Average Error per cent." for Localization of Tactile Stimuli on the Palmar and Dorsal Sets of Spots of each Finger on each of the Five Days immediately succeeding the First Attempt to Train the Palmar Spots of the Right Index Finger. Each Figure (except the Averages and Mean Variations in the Right-hand Columns) is calculated from Twenty-five Observations—save the Figures for the Palmar Set of Spots on the Trained Right Index Finger, which are calculated from Fifty Observations.

		Day of Experiment.						
Finger		9	10	11	12	13	Average	Mean variations
Palmar	L 3	0·0	0·0	3·3	13·9	0·0	= 3·44	4·18
	L 4	0·0	0·0	3·7	0·0	3·3	= 1·40	1·68
	R 2	**16·7**	**15·1**	**15·7**	**7·6**	**23·5**	**= 15·72**	**3·50**
	R 3	39·5	6·1	42·7	18·8	41·3	= 29·68	13·78
	R 4	41·8	39·8	42·0	43·0	48·7	= 43·06	2·23
	R 5	52·4	26·1	27·0	43·9	54·0	= 40·68	11·30
Dorsal	L 3	0·0	12·5	8·9	6·6	3·7	= 6·34	3·59
	L 4	0·0	0·0	11·1	16·9	0·0	= 5·60	6·72
	R 2	12·2	35·3	27·3	15·6	17·7	= 21·62	7·74
	R 3	7·5	0·0	14·7	19·4	37·0	= 15·72	9·98
	R 4	15·9	32·4	12·6	7·9	34·4	= 20·64	10·21
	R 5	14·4	28·3	28·0	11·1	29·0	= 22·16	7·53

The differences between these results and the corresponding ones which were taken before the "training" of the palmar spots of the right index finger will be discussed later. We may, however, note here that of the spots on the palmar aspects of the four fingers of the right hand those of the right index finger are now considerably the most accurately localized. In order of decreasing accuracy of localization of tactile stimuli these fingers are now arranged thus: R 2, R 3, R 5, R 4: whereas before the training of the right index finger ("R 2") that order was: R 3, R 2, R 5, R 4. But when the accuracy of localization is examined for the dorsal aspects of the same fingers they are arranged in order of decreasing accuracy of localization thus: R 3, R 4, R 2, R 5; that is, in exactly the same order as before the training.

When the figures for the palmar set of spots on the right index finger ("R 2"—the "trained" finger) are examined it is seen that there is a gradual decrease in average percentage error as the records proceed—except in the case of the test taken on the thirteenth day of the experiment. The conditions on that occasion were perhaps peculiar. The subject had spent a comparatively sleepless night, and on the following day he was too unwell to complete the tests. We have included this thirteenth record in our series, but the fourteenth is excluded as it is not a complete one.

In the next table (XV) the analysis of error of localization is given for the three individual spots in all the five records.

TABLE XV.—The Total Percentage Error of Localization for each of Spots "1," "2," and "3" on the Palmar and Dorsal Aspects of each of the Fingers examined on the Five Days which immediately followed upon the Training of the Palmar Set of Spots on the Right Index Finger. Each Percentage is calculated from about Forty-two Observations, except the Percentages for the Spots on the Palmar Aspect of the Right Index Finger (R_2) which are calculated from about Eighty-three.

		Spot		
Finger	1	2	3
Palmar L 3	0·00	4·35	6·82
L 4	0·00	4·55	0·00
R 2	**0·00**	**20·99**	**26·53**
R 3	14·29	35·42	40·48
R 4	15·38	42·50	69·57
R 5	39·53	41·46	36·59
Dorsal L 3	8·82	6·12	2·38
L 4	11·63	4·55	0·00
R 2	31·03	10·20	23·40
R 3	25·71	8·33	14·29
R 4	30·77	20·51	10·64
R 5	41·86	11·90	7·50

The number of observations from which the percentages are calculated in the case of the spots on the palmar surface of the right index finger ("trained") is double that in the case of the other fingers—i.e., a total of 250 observations for the three palmar spots of the right index finger as against 125 observations for the palmar spots of each of the other fingers, and for each of the dorsal sets of spots on all the fingers.

The most remarkable feature of this table is the entire absence of error of localization in the case of the spot on the palmar aspect of the terminal phalanx of the trained right index finger—that is, spot "1"; and the very great reduction of error in the case of the other two spots on the palmar aspect of that finger, as compared with the records before training. In the case of spot "1" no error of localization occurred at any of seventy-one observations distributed over the five different days. A comparison of this table with Table II is extremely instructive, but will be deferred to a later sub-section of this paper. It may, however, be pointed out here that in the case of the palmar sets of spots on the other three fingers there is a considerable similarity between the results obtained before and after the training. On the whole there is a small decrease in the error of localization of tactile stimuli; but if the order of accuracy is examined for the three palmar spots of each of the third, fourth, and fifth digits, it is found that it is the same before and after the training of the second digit—save only that in the case of the fifth digit, while spot "2" is still the least accurately localized, after the training spot "3" is the most accurately localized, while before it spot "1" was. More variation of the order of accuracy occurs in the case of the spots on the dorsal aspects of the fingers; but it will be observed that here, as before the training of the palmar set of spots on the right index finger, upon the whole spot "1" (that on the terminal phalanx) is the least accurately localized; while spot "3" (that on the basal phalanx) is the most accurately localized—the reverse of the findings in the case of the palmar spots.

It will be remembered that in the case of spot "3" on the palmar aspect of the fourth right digit, the error of localization was found to be greater than that which would have been expected to be given had the answers been selected by hazard. The error of hazard should here be 66·67 per cent.; and it will be seen that the error of localization for that spot on that finger is still greater than the hazard error, but yet not so great as before the training of the second digit. It is a matter of interest to examine this error again, and to compare it with the error for the same spot in the first series of records.

In Table III—column B—the percentages of error of localization for this spot on each of the five records taken after the training of the right index finger are given. And on Table IV—column B—there is an analysis of the total number of answers given for this spot in these records. When columns A and B are compared in that Table IV, it is seen that after the training (column B) the percentage of right localizations of that spot is about half as great again as the percentage before the training of the right index finger. On the other hand, the percentages for the wrong answers spot "1" and spot "2" are almost exactly the same as before. The increase in the number of right answers after the training of the other finger seems to have occurred at the expense of the "confused" answers which were given before the training. In other words, the subject now never says that he is unable to localize a tactile stimulus applied to this spot; but when he makes a wrong localization the percentage error for the wrong answer is the same as before. In this case the error of hazard would be 25 per cent.—or 33·33 per cent. if the answer "confused" be regarded as now eliminated from the possibilities. And yet the wrong answer spot "2" is given on 66·67 per cent. of occasions. There is, therefore, here a very definite "positive error" which cannot be explained by the mere taking away of a cortical function, but is rather to be looked upon as conditioned by a distortion of a cortical function. Into this question of "positive error" we shall inquire later.

In the records taken on the five days after the training of the right index finger, the state of "confusion" in answer to tactile stimuli practically disappeared. That is to say, that the subject scarcely ever indicated that he was unable to localize a stimulus.

In the following table (Table XVI) the figures for the localization by the subject of two consecutive similarly located stimuli upon two different spots are given as percentages of the number of times two such similar consecutive stimuli were applied. On comparison with the corresponding Table VI (for the figures before the training of the palmar spots on the right index finger) it will be seen that there is a considerable fall in the average error for spots on the palmar aspect of the right index finger; a considerably smaller fall in the case of these spots on the fourth right digit; little change in the case of the third right digit; and a considerable rise in that of the fifth. There is an even greater fall in this error in the case of the dorsal set of spots on the right index finger.

TABLE XVI.—The percentage number of times Pairs of Two consecutive similarly located Stimuli were Localized on Two Different Spots; given for each Aspect of each Finger on the Five Days which immediately succeeded the Training of the Palmar Set of Spots on the Right Index Finger, and expressed as Percentages of the number of times two similarly located Stimuli were given consecutively.

		Day of Experiment										Mean variation
	Finger	9	10	11	12	13	Average					
Palmar	L 3	0·0	0·0	16·7	0·0	0·0	=	3·3	..	5·3
	L 4	0·0	0·0	14·3	0·0	11·1	=	5·1	..	6·1
	R 2	**7·7**	**15·4**	**25·0**	**16·7**	**25·0**	=	**18·0**	..	**5·6**
	R 3	66·7	0·0	50·0	0·0	50·0	=	33·3	..	26·7
	R 4	20·0	0·0	50·0	0·0	50·0	=	24·0	..	20·8
	R 5	50·0	25·0	60·0	100·0	55·6	=	58·1	..	17·5
Dorsal	L 3	0·0	12·5	33·3	0·0	12·5	=	11·7	..	9·3
	L 4	0·0	0·0	0·0	0·0	0·0	=	0·0	..	0·0
	R 2	0·0	0·0	25·0	16·7	12·5	=	10·8	..	8·7
	R 3	0·0	0·0	66·7	16·7	75·0	=	31·7	..	31·3
	R 4	0·0	0·0	33·3	12·5	40·0	=	17·2	..	15·6
	R 5	14·3	12·5	20·0	16·7	66·7	=	26·0	..	16·2

In Table XVII the number of times two consecutive differently located spots were localized by the subject upon the same spot are given. A comparison with Table VII shows little change in the figures. Table XVIII gives the corresponding figures for cases in which the first of a pair of consecutive dissimilarly located tactile stimuli was rightly localized by the subject, but the second stimulus wrongly localized by him upon the same spot as the first. A comparison with the corresponding Table VIII (for the records before the training of the palmar spots on the right index finger) again shows little change after the training.

TABLE XVII.—The number of times two Different consecutive Stimuli were Localized upon the Same Spot—given for the Palmar and Dorsal Sets of Spots on each Finger on each of the Five Days which immediately succeeded the Training of the Palmar Set of Spots on the Right Index Finger.

		Day of Experiment										Mean variation
	Finger	9	10	11	12	13	Average					
Palmar	L 3	0	0	0	4	0	=	0·8	..	1·28
	L 4	0	0	1	0	0	=	0·2	..	0·32
	R 2	**5**	**5**	**3**	**1**	**6**	=	**4·0**	..	**1·60**
	R 3	7	2	7	1	10	=	5·4	..	3·12
	R 4	9	7	2	4	6	=	5·6	..	2·08
	R 5	5	4	2	7	6	=	4·8	..	1·44
Dorsal	L 3	0	1	0	2	1	=	0·8	..	0·64
	L 4	0	0	3	3	0	=	1·2	..	1·44
	R 2	4	7	2	2	3	=	3·6	..	1·52
	R 3	1	0	3	4	7	=	3·0	..	2·00
	R 4	5	5	2	2	8	=	4·4	..	1·92
	R 5	2	4	3	3	4	=	3·2	..	0·64

TABLE XVIII.—The number of times the Second of a Pair of Dissimilar consecutive Stimuli was Wrongly Localized on the same Spot as that on which the First of the Pair had been Correctly Localized—given for the Palmar and Dorsal Sets of Spots on each Finger on each of the Five Days which immediately succeeded the Training of the Palmar Set of Spots on the Right Index Finger. (In the case of each Record for each Finger, save that for the Palmar Aspect of the Right Index Finger—"R_2"—there were about Twenty Pairs of Dissimilar Stimuli given; in the case of "R_2" about Forty Pairs were given, and the number of Errors for that Finger has here been Divided by Two.)

		Day of Experiment.												Average		Mean variation
	Finger		9		10		11		12		13					
Palmar	L 3	..	0	..	0	..	0	..	2	..	0	=	0·4	..	0·64
	L 4	..	0	..	0	..	1	..	0	..	0	=	0·2	..	0·32
	R 2	..	**2·5**	..	**2·5**	..	**1·0**	..	**0·5**	..	**4·5**	=	**2·2**	..	**1·16**
	R 3	..	3	..	1	..	1	..	0	..	6	=	2·2	..	1·84
	R 4	..	5	..	4	..	1	..	2	..	3	=	3·0	..	1·20
	R 5	..	3	..	2	..	0	..	5	..	4	=	2·8	..	1·44
Dorsal	L 3	..	0	..	1	..	0	..	1	..	0	=	0·4	..	0·48
	L 4	..	0	..	0	..	2	..	2	..	0	=	0·8	..	0·96
	R 2	..	3	..	4	..	1	..	2	..	1	=	2·2	..	1·04
	R 3	..	1	..	0	..	2	..	2	..	5	=	2·0	..	1·20
	R 4	..	4	..	3	..	0	..	2	..	5	=	2·8	..	1·44
	R 5	..	2	..	3	..	2	..	2	..	3	=	2·4	..	0·48

A comparison of Tables XVI and XVIII seems to show that there is little or no regular relationship between the rates of incidence of these two types of error (one associated with defect of "individuality," the other with defect of "character") in the different fingers on the different days. Both errors may be relatively high in a certain finger, and relatively low in another; while, in yet another finger—or on another occasion—one error may be relatively high, and the other relatively low. But a comparison of the corresponding figures for these errors before the training of the palmar set of spots on the right index finger seems to hint that then there was an indication of a relationship between the two types of error. For when the incidence of one of the types of error is relatively high in a certain finger on a certain day, often the incidence of the other of the types of error in that finger on that day is relatively low (Tables VI, VIII).

(5) *On the Effect of the "training" of the Palmar Set of Spots on the right Index Finger: comparison of the Records before and after it.*

With regard to the effect of the training of the palmar sets of spots on the right index finger, three questions arise. In the first place, did an improvement in the accuracy of the localization of tactile stimuli upon these spots occur after the training, and was this improvement

markedly greater than any which may have occurred in the case of any of the other sets of spots examined in these experiments?

In the second place, provided such an improvement occurred, is it possible to refer it to any definite factors (such as, improvement in the "character" of the spots, improvement in their "individualities," "improvement in their positions"), and does this give us any information with regard to the initial state of badness of localization of tactile stimuli?

And, thirdly, what are the restrictions of the improvement, that is, can it be shown to occur in the case of sets of spots other than the trained ones, or is it confined to these?

Now, there is no doubt that a very definite and striking improvement in the accuracy of localization of tactile stimuli occurred in the case of the "trained" spots on the palmar aspect of the right index finger; and that no such great improvement occurred in the case of any other set of spots.

Table XIX sufficiently demonstrates this. In that table there is given side by side the average per record of the "average error per cent" of localization of tactile stimuli on the palmar and dorsal aspects of each of the fingers, in the first place before the training and in the second place after it.

TABLE XIX.—Comparison of the Errors of Localization on the different Fingers for: A, the Five Days immediately preceding; and B, the Five Days immediately succeeding the Training of the Spots on the Palmar Aspect of the Right Index Finger—expressed as the Average per Record of the "Average Errors per cent."; in Column C the difference between the two Averages is expressed as a Percentage of the First.

		A.		B.		C.
	Finger	Average	m.v.	Average	m.v.	Difference of averages
Palmar	L. 3	1·48	2·37	3·44	1·18	+ 1·96
	L. 4	4·52	5·42	1·40	1·68	− 3·12
	R. 2	42·70	3·80	**15·72**	**3·50**	− **26·98**
	R. 3	31·98	8·30	29·68	13·78	− 2·30
	R. 4	46·72	5·96	43·06	2·23	− 3·66
	R. 5	43·06	6·37	40·68	11·30	− 2·38
Dorsal	L. 3	0·66	1·06	6·34	3·59	+ 6·34
	L. 4	2·04	1·63	5·60	6·72	+ 3·56
	R. 2	28·56	7·19	21·62	7·74	− 6·94
	R. 3	18·76	2·94	15·72	9·98	− 2·04
	R. 4	26·80	8·40	20·64	10·21	− 6·16
	R. 5	32·62	9·22	22·16	7·53	− 10·46

It will be seen that there is a very marked fall (from 42·70 per cent. to 15·72 per cent.) in the error for the palmar spots of the right index

finger ("R 2") after the training of them. The difference of these two percentages is about 27; and, although there is a slight improvement in the case of the spots on the palmar aspects of the other fingers of the right hand, the difference of the percentages of the finger which shows the greatest improvement after the index finger (the fourth digit) is less than 4. Reckoned as percentages of the larger percentage (that before the training) these differences would be about 63·2 per cent. and 7·8 per cent. respectively. This therefore demonstrates a very great improvement of localization of tactile stimuli on the palmar set of spots of the right index finger, and a slight improvement on these spots of the other fingers of the right hand.

When the figures for the sets of spots on the dorsal aspects of the right fingers are examined, it is seen that in every case an improvement of localization of tactile stimuli has occurred. The greatest improvement occurs in the case of the little finger, but the difference between the two percentages there is 10—about 32 per cent. of the figure before the training of the palmar spots on the index finger.

A great improvement of the localization of tactile stimuli on the three palmar spots of the right index finger therefore occurred—in what did it consist? It is possible to examine here the error for each of the three spots separately, and to examine the kinds of error which were made before and after the training.

Now with regard to the error for each of the three spots—in the following table (Table XX) the total number of times the four possible answers were given in reply to tactile stimuli, applied to each spot of each aspect of the fingers of the right hand, is expressed as a percentage of the total number of times each spot was touched in the tests before and after the training of the palmar set of spots on the right index finger.

Obviously, if the subject knows that the spot touched is one of three possible ones there are four possible answers to each tactile stimulus—that is, "It is on spot 1," spot "2," or spot "3," while the fourth answer is "I cannot decide"—which we may call "confusion." Of the answers spot "1," spot "2," and spot "3" one is right for each spot the tactile stimulus is located upon; in the table the figures for the right answer to each spot are emphasized.

We notice at once that in the case of the right index finger (palmar aspect), whereas the localization of spot "1" was right on 66·67 per cent. of occasions before the training, after the training the percentage rises to 100—no mistakes were made. The difference between these

percentages is about 33. When we examine the figures for the corresponding spot ("1"—the spot on the terminal phalanx) on the palmar aspects of the other fingers of the right hand, and on the dorsal aspects of all the right fingers, we find that in the case of this spot on the palmar aspect of the fourth digit and on the dorsal aspects of the second and fourth digits there is also an improvement. But it is comparatively slight—the difference of the percentages in the case of the greatest of these improvements being about 15. In all the other cases the localization of tactile stimuli upon spot "1" is worse after the training of the right index finger.

TABLE XX.—The Average Percentage per Record of the different Answers ("Spot 1," "Spot 2," "Spot 3," and "Confused") given to the Tactile Stimuli applied to each of the Three Spots on each aspect of each Finger: A, in the Five Records before the Training of the Palmar Set of Spots on the Right Index-finger; and B, in the Five Records after that Training.

			A. Before. Answers				B. After. Answers			
Finger		Spot	"1"	"2"	"3"	"Con."	"1"	"2"	"3"	"Con."
Palmar	R. 2	*1*	**67·57**	*18·92*	*5·41*	*8·11*	**100·00**	*0·00*	*0·00*	*0·00*
		2	*10·42*	**37·50**	*27·08*	*25·00*	*2·47*	**79·01**	*18·51*	*0·00*
		3	*5·00*	*20·00*	**65·00**	*10·00*	*2·04*	*24·49*	**73·47**	*0·00*
	R. 3	1	**86·67**	8·89	0·00	4·44	**85·71**	11·43	2·86	0·00
		2	7·89	**73·68**	7·89	10·53	14·58	**64·58**	20·83	0·00
		3	4·76	47·62	**40·47**	7·14	4·76	35·71	**59·52**	0·00
	R. 4	1	**74·29**	14·29	2·86	8·57	**84·62**	7·69	7·69	0·00
		2	7·84	**68·63**	11·76	11·76	10·00	**57·50**	30·00	2·50
		3	2·56	66·67	**20·51**	10·26	4·35	65·22	**30·43**	0·00
	R. 5	1	**62·22**	17·78	15·56	4·44	**60·46**	34·88	4·65	0·00
		2	2·22	**53·33**	28·89	15·56	21·95	**58·54**	19·51	0·00
		3	5·71	31·43	**60·00**	2·86	0·00	34·15	**63·41**	2·44
Dorsal	R. 2	1	**54·05**	18·92	5·41	21·62	**68·97**	27·59	3·45	0·00
		2	0·00	**75·00**	18·75	6·25	6·12	**89·80**	4·08	0·00
		3	0·00	7·50	**85·00**	7·50	2·13	21·28	**76·60**	0·00
	R. 3	1	**84·44**	2·22	0·00	3·33	**74·29**	20·00	5·71	0·00
		2	5·26	**65·79**	15·79	13·16	4·17	**91·67**	4·17	0·00
		3	2·38	4·76	**92·86**	0·00	0·00	14·29	**85·71**	0·00
	R. 4	1	**68·57**	8·57	5·71	17·14	**69·23**	28·21	2·56	0·00
		2	9·80	**68·63**	13·73	7·84	7·69	**79·49**	12·82	0·00
		3	7·69	10·26	**79·49**	2·56	4·26	6·38	**89·36**	0·00
	R. 5	1	**62·22**	17·78	6·67	13·33	**58·14**	30·23	11·63	0·00
		2	20·00	**60·00**	4·44	15·56	4·76	**88·10**	7·14	0·00
		3	2·86	5·71	**82·86**	8·57	5·00	2·50	**92·50**	0·00

In the case of spot "2" (that on the middle phalanx) a yet greater relative improvement occurs on the right index finger—but error here does not disappear after the training. In the case of no other finger does improvement of localization of the corresponding spot proceed to

such a great extent; and in the case of the palmar aspects of the fingers other than the index finger improvement occurs only in the case of spot "2" on the fifth digit—and there the difference in the percentages is only about 5, whereas in the case of the index finger it is about 42.

In the case of the localization on spot "3" of the palmar aspect of the right index finger (the spot on the basal phalanx) a slight improvement occurs. A similar improvement also takes place in the case of the corresponding spot on the palmar aspects of the third, fourth, and fifth digits (in the case of the third and fourth digits being much greater than the improvement in the case of the trained index finger) and on the dorsal aspects of the fourth and fifth digits (the improvement there being also slightly greater than that in the case of the palmar aspect of the right index finger); the localization of spot "3" on the dorsal aspects of the right second and third digits is worse after the training of the palmar set of spots on the index finger.

It may perhaps be inferred that the improvement of the accuracy of localization of tactile stimuli upon spot "3" on the palmar and dorsal aspects of all the fingers is a more or less general one; but that the great improvement of localization on spots "1" and "2" on the palmar aspect of the right index finger is a special one conditioned by the training given to that set of spots on this finger.

When the answers for the palmar aspects of the right index finger are examined, it is seen at once that "confusion" disappears after the training. In the case of tactile stimuli applied to spot "1" the wrong answers spot "2" and spot "3" also disappear; but in the case of spot "2"—while "confusion" does not occur after the training—the wrong answers spot "1" and spot "3" are only reduced. The reduction is, however, large and the increase in the accuracy of the localization of stimuli applied to this spot may perhaps be looked upon as conditioned by the absence of confusion—the decision now being made in the right direction, and by the reduction of the wrong replies. When the answers to spot "3" are examined it is seen that the wrong reply spot "1" is given slightly less often after the training, and the wrong reply spot "2" slightly more often. These differences are small, and the increase in the proportion of right answers may perhaps be looked upon as conditioned by the absence of confusion—as it were the decision being now made, and in the right direction. It will be seen that "confusion" practically disappears for all the fingers after the training of the right index finger; but it is by no means the case that the

improvement of the accuracy of localization of a spot is greatest where the proportion of "confusion" answers was greatest before the training of the right index finger.

An interesting point is exhibited by this table. Where, before the training of the right index finger, the proportion of confusion answers is great for all the fingers the proportion of each answer to each spot should have been about 25 per cent. had the answers been selected by hazard. After the training of the index finger it is perhaps fairer to assume that, as "confusion" practically disappears for all the fingers, the hazard proportion should have been about 33·3 per cent. for each answer. Now, when the table is examined, it is seen that in the case of several answers the proportion is greater than that of hazard. Except in the case of the right answer to spot "3" on the palmar aspect of the fourth right digit both before and after training of the index finger, this is uniformly so both before and after the training for the correct answers to each spot on each aspect of each finger. In other words, there is almost uniformly a positive bias in the direction of the correct answer. But when the incorrect answers are examined it is seen that in certain cases there is an error greater than the error of hazard. (Finger R 2—answer spot "3" to stimuli on spot "2"; R 3—spot "2" to spot "3"; R 4—spot "2" to spot "3"; R 5—spot "3" to spot "2," and spot "2" to spot "3"—all on the palmar aspects of the fingers, no instance on the dorsal, before training; and R 3—answer spot "2" to stimuli on spot "3"; R 4—spot "3" to spot "2," and spot "2" to spot "3"; R 5 spot "1," spot "2" to spot "3"—all on the palmar aspects of the fingers after training, and, perhaps, R 2—answer spot "2" to stimuli on spot "1"; R 4—spot "2" to spot "1"; R 5—spot "2" to spot "1"—all on the dorsal aspects of the fingers after training of the spots on the palmar aspect of the index-finger). In the case of stimuli applied to spots "2" and "3" on the palmar aspects of the fingers there seems to be a fairly definite incorrect identification of "2" with "3" and "3" with "2." In the case of the dorsal sets of spots there is, after the training of the palmar set of spots on the right index finger, a most curious increase of the mistake of localizing stimuli which were applied to spot "1" upon spot "2"—and this is the case for all the fingers. After the training of the palmar spots of the index finger this mistake becomes positive—greater than the error of hazard.

Before we leave this table it may be noticed that in the case of the answer spot "1" this error diminishes for spots "2" and "3" on the

palmar aspect of the right index finger (trained) along with the increase of accuracy of localizing stimuli applied to spot "1" itself; but there is no corresponding decrease in this error for spots "2" and "3" in the case of the other fingers (except perhaps in the case of spot "3" on the little finger) and no corresponding increase of accuracy of the localization of spot "1" on these fingers. From this it would appear that the effect of the training is not spread over the other fingers—but we shall return to this question later.

In Table XXI the average number of times two consecutive similarly situated stimuli were localized upon different spots is given for the records before and after the training of the palmar spots on the right index finger. It will be seen that a considerable decrease in this form of error took place in the case of the palmar and dorsal sets of spots on the right index finger; that a considerable decrease took place for the dorsal set of spots on the little finger; and that a great increase occurred in the case of the palmar spots of the little finger. In the other instances the variation is probably negligible.

TABLE XXI.—Comparison of the Average number of times per. cent. per Record that Pairs of Two consecutive similarly situated Stimuli were Localized upon two different Spots: A, Before the Training of the Palmar Set of Spots on the Right Index Finger; and B, After that Training.

		A		B		
Finger		Average	m.v.	Average	m.v.	Differences of averages
Palmar	L. 3	8·0	12·8	3·3	5·3	− 4·7
	L. 4	5·0	8·0	5·1	6·1	+ 0·1
	R. 2	40·0	16·0	**18·0**	**5·6**	− **22·0**
	R. 3	34·5	19·6	33·3	26·7	− 1·2
	R. 4	32·3	7·9	24·0	20·8	− 8·0
	R. 5	28·3	12·8	58·1	17·5	+ 29.8
Dorsal	L. 3	2·9	4·6	11·7	9·3	+ 8·8
	L. 4	7·5	9·0	0·0	0·0	− 7·5
	R. 2	38·0	17·6	10·8	8·7	− 27·2
	R. 3	23·8	12·4	31·7	31·3	+ 7·9
	R. 4	25·5	1·37	17·2	15·6	− 8·3
	R. 5	48·3	24·2	26·0	16·2	− 22·3

The chief point of interest is that here the improvement of this error in the case of the right index finger applies not only to the trained spots on the palmar aspect but also to the untrained ones on the dorsal aspect.

In Tables XXII and XXIII the errors in which the same answer was given to two consecutive stimuli which were situated on different spots are compared for the records before and after the training of the palmar spots on the right index finger—in the second table the figures apply

only to those errors in which the first spot was correctly localized and the second incorrectly, on the same spot as the first. It is seen that, although this error decreases for the palmar spots of the right index finger, the decrease is not so great as that in the case of some of the other fingers.

TABLE XXII.—Comparison of the Average number of times per Record two different consecutive Stimuli were Localized upon the same Spot: A, Before the Training of the Palmar Set of Spots on the Right Index Finger; and B, After that Training.

		A		B		
	Finger	Average number	m.v.	Average number	m.v.	Difference of averages
Palmar	L. 3	0·0	0·0	0·8	1·28	+ 0·8
	L. 4	0·8	0·96	0·2	0·32	− 0·6
	R. 2	5·4	2·5	**4·0**	**1·60**	− **1·4**
	R. 3	4·8	1·4	5·4	3·12	+ 0·6
	R. 4	5·6	2·1	5·6	2·08	.. 0·0
	R. 5	6·8	1·8	4·8	1·44	− 2·0
Dorsal	L. 3	0·0	0·0	0·8	0·64	+ 0·8
	L. 4	0·8	0·6	1·2	1·44	+ 0·4
	R. 2	3·8	0·6	3·6	1·52	− 0·2
	R. 3	3·4	1·9	3·0	2·00	− 0·4
	R. 4	4·2	1·5	4·4	1·92	+ 0·2
	R. 5	4·4	1.1	3·2	0·64	− 1·2

TABLE XXIII.—Comparison of the Average number of times per Record the Second of a Pair of consecutive dissimilar Stimuli was Wrongly Localized upon the same Spot as that on which the First Stimulus had been Correctly Localized: A, Before the Training of the Palmar Set of Spots on the Right Index Finger; and B, After that Training.

		A		B		
	Finger	Average number	m.v.	Average number	m.v.	Difference of averages
	L. 3	0·4	0·64	0·4	0·64	.. 0·0
	L. 4	0·2	0·32	0·2	0·32	.. 0·0
	R. 2	2·4	0·96	**2·2**	**1·16**	− **0·2**
	R. 3	2·8	0·64	2·2	1·84	− 0·6
	R. 4	2·6	1·52	3·0	1·20	+ 0·4
	R. 5	3·6	1·12	2·8	1·44	− 0·8
Dorsal	L. 3	0·0	0·00	0·4	0·48	+ 0·4
	L. 4	0·6	0·48	0·8	0·96	+ 0·2
	R. 2	2·0	0·80	2·2	1·04	+ 0·2
	R. 3	1·8	1·04	2·0	1·20	+ 0·2
	R. 4	2·0	0·80	2·8	1·44	+ 0·8
	R. 5	2·6	0·88	2·4	0·48	− 0·2

It is interesting to compare Table XXIII with Table XXI. It is then seen that there is little correspondence between the two kinds of error. For a set of spots on one finger both errors may fall after the training; or both may rise; or one may rise and the other may fall.

Before we pass to the consideration of these results one more analysis may be given—that of the occurrence of "dilemma" before and after the training of the index finger. The observation of this phenomena depends very largely upon the experimenter, and the figures here are given with this reservation—for it is extremely difficult to determine in what the dilemma consists. In our experiments the subject pointed with a pencil to the spot on the hand of the model which he supposed to correspond with the spot on his own hand which had just been touched. In doing this he usually placed the point of the pencil directly upon the spot which he wished to indicate. Occasionally the latency of this act was obviously much greater than usual; and then we sometimes counted the answer as one which exhibited "dilemma." But more usually there was a very distinct indication of the presence of dilemma. The pointer would hover over the spots on the hand of the model—now over one, now over another—and finally the subject would either place the pointer upon one of the spots (in which case we called the answer one characterized by dilemma), or would touch the table in indication that he was unable to locate the tactile stimulus (in which case we called the answer "confusion"). In all these records no word was spoken from beginning to end, and our difficulty was to distinguish "dilemma" from mere delay of response where the pointer did not hover between different spots, and to notice the small indications of this hovering which sometimes occurred. One very marked change occurred after the training of the palmar set of spots on the right index finger, but we had no instruments to record it. The latency of the localizing act of the subject was always very much greater for stimuli applied to the right hand than for those applied to the normal left hand. After the training of the palmar set of spots on the right index finger the latency of their localization was markedly less than that of the localization of tactile stimuli upon the palmar spots of the other fingers; but it never became as small as that of the localization of spots on the left hand.

In the following table (Table XXIV) the number of times dilemma occurred in the five records before the training of the right index finger and in the five records after that training is given. A distinction is drawn between cases of dilemma in which the final answer was wrong and cases in which it was right.

The decrease of this phenomenon after the training of the palmar spots on the right index finger is seen to occur generally for all the sets of spots examined, and not to be greater in the case of the index finger than in the cases of the other fingers. We do not give a more minute

analysis, for our figures give no indication of a relation between the number of times dilemma occurred and the relative accuracy of localization either for the set of spots on a finger or for any one of these spots.

TABLE XXIV.—A comparison of the number of times "Dilemma" occurred: A, in the Records Before the Training of the Palmar Set of Spots on the Right Index Finger; and B, in the Records, After that Training. In the columns the First number denotes the times the Final Result of the Dilemma was Wrong, the Second number the times the Final Result was Right, and the Third gives the Total of the Two.

		A Before			B After		
Finger		Wrong	Right	Total	Wrong	Right	Total
Palmar	L 3	0	0	0	0	0	0
	L 4	0	1	1	0	0	0
	R 2	3	11	14	**3**	**5·5**	**8·5**
	R 3	3	16	19	0	7	7
	R 4	4	12	16	4	5	9
	R 5	6	9	15	3	2	5
Dorsal	L 3	0	0	0	0	0	0
	L 4	1	1	2	0	4	4
	R 2	5	15	20	2	6	8
	R 3	3	14	17	0	5	5
	R 4	3	22	25	0	6	6
	R 5	1	17	18	0	7	7

We may now summarize the results which were obtained after the training of the palmar set of spots on the right index finger.

In the first place a general improvement of the localization of tactile stimuli occurred for all the sets of spots examined in this experiment. But a much greater improvement occurred in the case of the trained set of spots than in the case of any other set, and we are probably justified in attributing the improvement to the training.

This improvement in the case of the trained palmar set of spots was most marked in the case of spot "1" (on the terminal phalanx) and spot "2" (on the middle phalanx). In the localization of the first of these all error disappeared after the training; in the localization of the second each type of error markedly decreased. In the localization of spot "3" (basal phalanx) little change occurred in the different types of error, save that "confusion" disappeared.

Now this improvement might (on our hypothesis) be due (1) to an improvement in the attribute of character of the trained spots (whereby they are recognized as different amongst themselves); or (2) to an improvement of the attribute of individuality (whereby stimuli applied to a specific spot are recognized as similarly located); or (3) to an

improvement of the attribute of position (whereby the tactile stimulus is referred to a special spot on the surface of the body).

In the early records of the experiment—those taken before the training of the right index finger—there was a great tendency for the answers to run in consecutive series, although the stimuli were applied indifferently to the different spots. This is what would be expected to occur if the attribute of character of the tactile stimuli was lost, or at any rate impaired.

In the training which we gave to the palmar spots on the right index finger, the subject in one part of it especially concentrated his attention on the difference between the tactile stimuli which he could then see being applied to the different spots in an indifferent order.

TABLE XXV.—A comparison of the total number of times Series of consecutive similar Answers of different lengths were given: A, in the Records Before the Training of the Right Index Finger; and B, After that Training; the Second columns give Arbitrary Figures obtained by Multiplying the Number of Series in the First columns by the Number of Answers in the Series (2, 3, 4, &c.). The Figures for the Dorsal Sets of Spots are not given.

	A. Before				B. After			
Finger	No. *in* series	No. *of* series			No. *of* series			
R 2	2	7	14		7		14	
	3	7	21		3		9	
	4				1		4	
	5	1	5		1		5	
	6							
	7							
	8				0·5		4	
	9	1 = 16	9 = **49**		= 12·5		= **36**	
R 3	2	7	14		11		22	
	3	5	15		1		3	
	4	1	4		3		12	
	5	1	5		2		10	
	6	1 = 15	6 = **44**		1 = 18		6 = **53**	
R 4	2	3	6		11		22	
	3	3	9		5		15	
	4	3	12		5		20	
	5	4	20		1		5	
	6				1		6	
	7	1 = 14	7 = **54**		= 23		= **68**	
R 5	2	14	28		13		26	
	3	3	9		6		18	
	4	1	4		2		8	
	5	3	15					
	6	2 = 23	12 = **68**		1 = 22		6 = **58**	

After the training the records show a reduction in the number of such consecutive series of similar answers for the spots on the palmar aspectof the right index finger. In the cases of the third and

fourth right digits an increase in this number occurred—that increase being a considerable one in the case of the fourth. While the number in the case of the fifth digit was practically stationary. The figures are given in Table XXV.

This would seem to demonstrate that one of the initial factors in the subject's badness of localization of tactile stimuli consisted in the impairment of the attribute of "character" of the tactile stimuli—the attribute whereby the subject is conscious that tactile stimuli located upon different spots are, in some manner, dissimilar—and that the effect of the training of the spots on the right index finger was to reduce this error for them, but not (at any rate to the same extent) to reduce this error for the spots on the other fingers. It must, however, be noticed that when these figures are analysed in terms of pairs of dissimilarly located stimuli, the first of which is correctly localized by the subject and the second wrongly upon the same spot as the first, the error is not more greatly diminished for the index fingers than for the others (Table XXIII)—but this is perhaps only a part of the special type of error which we are examining.

It would, therefore, seem that impairment of the attribute of character was one of the factors in the initial condition of the subject's inaccuracy of localization of tactile stimuli, and that this impairment was reduced by training a specific set of spots. But it must be noted that the hypothetical attribute of character may not be eliminated—only impaired; for even in the earliest records there were portions where the answers did not run in series, and where at times the localization of tactile stimuli was accurate.

When the early records are examined it is found that at times series of consecutive similarly located stimuli were localized by the subject upon different spots. At these times the answers, of course, did not run in similar series; and the inference (on our hypothesis) is that this error was conditioned by impairment of the attribute of "individuality" in the localization of tactile stimuli—the attribute whereby the subject knows that tactile stimuli applied to the same spot are in some manner similar to each other.

In a part of our training the subject paid special attention to the similarity between tactile stimuli which he saw being applied in consecutive series to a single spot.

After that training the records show a decrease in this type of error—the localizing of consecutive similarly located stimuli upon dissimilar spots—in the case of the trained spots. But there is also a

decrease in the case of the untrained spots on the back of the trained finger. From this it might be inferred that this attitude is, as it were, common to various sets of spots on a finger; but in the case of the little finger a marked increase of error took place for the palmar set of spots, and a marked decrease for the dorsal, after the training of the index finger. When the palmar sets of spots on the fingers are alone considered, the decrease in this type of error is seen to be much less in the cases of the third and fourth digits than in the case of the trained index finger; while an increase of error occurs in the case of the fifth digit (Table XXI).

It may, perhaps, be inferred that this error decreased after the training of the palmar set of spots on the right index finger for that finger, and not to so great an extent for the palmar sets of spots of the other fingers of the right hand; that this error is conditioned by impairment of the attribute of individuality in the localization of tactile stimuli; and that the training reduced this impairment.

If so, we may regard the original condition of the subject as conditioned in part by impairment of the attribute of character, and in part by impairment of the attribute of individuality.

But for the complete and accurate localization of tactile stimuli there is necessary (on our hypothesis) the attribute of "position." In the subject whose state we examined this attribute can hardly have been lost; for then it would be expected that he would have been unable to localize the tactile stimuli; whereas one of the features of this case was the comparatively small proportion of instances in which he could not locate a stimulus. But there seems to be evidence for a definite distortion of this attribute.

That this is so is shown by the occurrence of "positive error" in the localization of certain spots. The error which he made in the localization of certain spots was greater than the error of hazard. In such a case the subject had three choices (excluding the possible "confusion" answer). One of these was the correct answer, and the other two were incorrect. Now, when these errors are analysed (Table XX, see analysis in text), it is seen that in some cases a certain wrong answer would be given more often than the error of hazard. In such cases a distortion, probably of the attribute of position, must be assumed. That is to say, that in certain instances the subject showed a definite tendency to place a certain specific tactile stimulus upon a certain wrong spot rather than upon a certain other wrong spot. The "position" of the tactile stimulus had in some manner been distorted.

This occurred in the case of one of the wrong answers given to spot "2" on the palmar aspect of the right index finger. That was one of the two trained spots which most definitely showed improvement of localization after the training. In the records taken after the training, this positive error was reduced considerably below the error of hazard, but this reduction was not accompanied by an increase on the other possible wrong localization of this spot. In one of the other cases of positive error which occurred in the other fingers before the training of the right index finger, a reduction below the error of hazard also occurred; but in this case that reduction was accompanied by a marked increase in the other possible error in the localization of that spot.

It may, perhaps, be assumed that part of the initial condition of this subject's inaccuracy of localization of tactile stimuli was due to a distortion of the attribute of position; and that after the training this distortion was reduced for the spots which were trained.

One further point with regard to the analysis of the effects of the training—the question whether it was confined to the trained spots or was seen also in others—may be left over to the following subsection of this paper.

(6) *On the Question whether the Improvement in Accuracy of Localization after Training was confined to the Spots which were trained.*

To a certain extent this question may at once be answered. An apparent general improvement of all the spots on the back and front of the fingers occurred after the training of the palmar set of spots on the right index finger. This apparent general improvement was, however, much less than the special improvement of the trained set of spots.

Now, although such an apparent general improvement took place, it cannot definitely be ascribed to the training of a special set of spots. Thus it is, perhaps, possible that the mere repetition of the test might be accompanied by a general improvement in the subject's responses—even although he was never allowed to know when his replies were right or when they were wrong.

To test the possible general effect of the training we used the following method: A second set of spots was selected on the palmar aspect of each finger. These spots lay on the folds of skin which are situated near the inter-phalangeal and metacarpal-phalangeal joints—whereas the spots of the sets which we used in the daily examinations lay over the middle of the phalanges. The set of spots which were

examined from day to day (and were trained on the right index finger) we may term the "ordinary spots" or the "trained spots"; while the other set of spots, which was examined, but rarely so that there might be little or no effect of practice, we may term the "untrained spots," because no set of them were trained.

We had not thought of selecting these second sets of spots before the commencement of the training of the spots on the right index finger, and so we are unable to compare the average errors for the two sets of spots on each finger before the records were complicated by the training. It is possible that the errors were not the same (or not very nearly the same) for the two sets of spots. In another case, which was in almost every respect similar to that which we now describe, we found that the error of localization of tactile stimuli was distinctly greater for the sets of spots over the joints than for the sets of spots which were placed over the middle of the phalanges—although in this case the subject thought that he could localize the former spots more accurately than the latter. When the localization is carried out in an exactly similar manner on the ordinary subject there is little or no difference in the accuracy of localization of supra-liminal tactile stimuli on these two sets of spots—at any rate, with the instrument which we used.

After the training of the palmar set of "ordinary spots" on the right index finger we examined on three occasions the accuracy of localization for the "untrained spots" on the fingers. Two of these examinations were done on the twelfth day of the experiment, and the third on the thirteenth. The following table gives the average error per cent. of localization:—

TABLE XXVI.—Average Error per cent. of Localization on the "Untrained" Sets of Spots on the Palmar Aspects of the Fingers after the Training of the "Ordinary" Set of Spots on the Right Index Finger.

	Day of Experiment						
Finger	12		12a		13		Average
Palmar L 3 ..	18·0	..	4·8	..	0·0	=	7·6
L 4 ..	13.7	..	3·3	..	7·0	=	8·0
R 2 ..	**60·0**	..	**65·3**	..	**48·1**	=	**57·8**
R 4 ..	37·6	..	35·2	..	47·3	=	40·0
R 3 ..	28·7	..	55·6	..	41·2	=	41·8
R 5 ..	54·1	..	27·6	..	52·9	=	44·9

A glance at this table shows that the index finger ("R. 2") is actually the worst of the four fingers of the right hand. That is to say that this finger—which at this time, after the training, was the best of the four

as regards the localization of tactile stimuli on the "ordinary" spots—is much the worst for the localization of these stimuli when a strange set of spots is selected for examination. The figures in columns 12 and 13 of this table should be compared with those in the corresponding columns (12 and 13) of Table XIV. The latter figures are those for the "ordinary" spots on the same day. Especially when the figures in the column 13 are examined, it will be seen that there is considerable similarity between them as regards the third, fourth, and fifth digits—but that there is a considerable difference when the figures for the second digit (index finger) are examined. The percentage of error is 23·5 for the "ordinary" spots on that finger, and 48·1 for the "untrained" spots; a still greater difference (7·6 per cent. as against 60 per cent.) occurs in column 12. The column of averages may be compared with those in Table XIX ("ordinary" spots, before and after training of right index finger).

TABLE XXVII.—The Total Percentage Error of Localization for each of Spots 1, 2, and 3 of the "Untrained" Sets on the Palmar Aspects of the Fingers in the Three Records given in Table XXVI.

	Spot		
Finger	1	2	3
Palmar L 3 ..	14·9 ..	3·7 ..	0·0
L 4 ..	7·4 ..	16·7 ..	0·0
R 2 ..	**73·3 ..**	**50·0 ..**	**50·0**
R 3 ..	66·7 ..	42·4 ..	11·1
R 4 ..	45·8 ..	29·6 ..	50·0
R 5 ..	13·9 ..	66·1 ..	51·2

The next table (Table XXVII) gives the average error of localization for each of the three "untrained" spots on each finger in these three records. The corresponding figures for the "ordinary" spots before training the right index finger are given in Table II, and those after the training in Table XV. A comparison of these two tables with the present one demonstrates scarce a point of resemblance. The incidence of error as regards the distal, intermediate, and proximal spots (spots "1," "2," and "3") seems to have no common basis when the figures for the "ordinary" and "untrained" sets of spots are examined. This strongly suggests that the different sets of spots on the same aspect of the same finger have little or nothing in common. On comparison of the figures for spot "1" on the right index finger in this table and in Table XV it will be observed that in the latter case, after training, error has absolutely disappeared; but in the former case (that is, an "untrained" spot which lies in a corresponding position in its set) the

error is as great as 73·3 per cent., and is greater than that of either of the other spots in the set.

The conclusion from these figures would seem to be that the training of a set of spots on a certain finger, although very definitely reducing the error in the localization of tactile stimuli for the trained set of spots, does not reduce the error for other and untrained sets of spots on the same finger. Arguing from this alone, it might be said that the effect of training is confined to the trained spots, and is extended neither to other spots on the trained finger nor to spots on the other fingers. The apparent increase of accuracy in the localization of tactile stimuli on the "ordinary" spots of the other fingers (as shown by our experiments) might then be supposed to be conditioned merely by the effect of practice.

But in this connection it must be noted that the spots which exhibited an increase of accuracy of localization on the other fingers lay in corresponding positions to the trained spots on the right index finger. It might be that the training of the spots on the right index finger was accompanied by a slight increase in accuracy of localization for the corresponding spots on the other fingers—but not by an increase in accuracy for the differently lying spots either on the trained finger or on the other fingers. An examination of the figures in Table XX shows, however, that the increase in the accuracy of localization in the "ordinary" spots of the untrained fingers does not take place to any appreciable extent in the spots ("1" and "2") which correspond to those which exhibit a marked increase of accuracy of localization in the case of the trained finger; but occurs chiefly in a spot ("3") which on the trained finger exhibits comparatively small increase of accuracy. We are, therefore, probably correct in ascribing the general increase in the accuracy of localization on all the fingers to a general process—perhaps to that associated with "practice."

Another point of interest arises in connection with a possible extension of the effect of the training of the spots on a single finger. In each of the records examined so far, the subject was allowed to know which finger was being stimulated at any one time. Now, for accurate localization of a tactile stimulus, the subject must be aware in the first place of the part of the body which is touched, and in the second place of the place of the spot which is touched upon that part. The part of the body is, of course, a relatively ill-defined concept in the case of most subjects; the subject may have difficulty in defining where the wrist ends and the forearm begins. But, for our purposes, it is perhaps

sufficient to suppose that "front of finger" (or merely "finger") and "place on finger," were two elements in the "position" of the spots which we were examining. Now, error in the first of these elements was eliminated for most of our records by allowing the subject to know which finger was being touched.

But as the experiment proceeded there occurred a definite increase in the accuracy of localization of tactile stimuli on the trained spots of the trained finger. The interesting question arose—does the training of a set of spots on a finger under these conditions enable the subject more accurately to locate tactile stimuli upon that finger than upon the others when he is not allowed to know which finger is being touched? In other words, did the increase of accuracy of localization extend to the element "finger" as well as to the element "place on finger" in the "position" of spots on the trained finger, or was it confined to the element "place on finger"?

To examine this point we used a test in which the "ordinary" spots were touched upon the different fingers in an indifferent order—so that no one finger was often touched twice in succession.

The analysis of such a test is one of difficulty. Here we confine ourselves to the examination of the errors in placing the spot touched irrespective of the fingers, and in localizing the fingers touched irrespective of error in the placing of the spot. Obviously, the finger may be right and the spot on it may be wrongly placed; or the spot may be correctly placed, but upon the wrong finger; or both finger and place on finger may be correctly localized, or both may be wrong. We must refer to a point which touches the accuracy of our results. These tests were comparatively short ones. Except in the case of the last in this series, an average of a little more than 6 stimuli were applied to each finger in each test, and of a little more than 8 to each position of the three spots. In the last test about 50 stimuli were given to each finger, and about 67 to each position of the three spots—that is, about 16·7 stimuli to each individual spot.

We began this test—which may be termed the "unknown finger" test—the day before the training of the "ordinary" spots on the palmar aspect of the right index finger; and continued it, with one exception, every day thereafter. In Tables XXVIII and XXIX the figures are given for the record taken before the commencement of the training of the right index finger and for the two records taken immediately after the commencement of it.

In Table XXVIII the percentage error of localization of each indi-

vidual spot is given without reference to the rightness or wrongness of the finger upon which it was localized. The averages for each of these three spots are rather larger than the corresponding ones for the first five records in this experiment (obtained from Table II). Thus: "1" = 20·5 per cent.; "2" = 30·5 per cent.; "3" = 49·4 per cent.; average of the three = 33·5 per cent. Whereas in the first five records, taken before the training of the right index finger was commenced, these averages were: "1" = 27·3 per cent.; "2" = 41·7 per cent.; "3" = 53·5 per cent.; average of the three = 40·8 per. cent.

TABLE XXVIII.—THE PERCENTAGE ERROR OF LOCALIZATION OF THE INDIVIDUAL SPOTS, "1," "2," AND "3," IN THE "UNKNOWN FINGER" TEST UPON THREE SEPARATE DAYS: THE FIRST BEFORE TRAINING OF THE PALMAR SET OF SPOTS ON THE RIGHT INDEX FINGER, THE SECOND ON THE DAY AFTER THE COMMENCEMENT OF THAT TRAINING, THE THIRD ON THE SECOND DAY. IMMEDIATELY UNDERNEATH THESE PERCENTAGES IS GIVEN THE AVERAGE OF THE THREE PERCENTAGE ERRORS; BELOW THAT IS GIVEN THE AVERAGE PERCENTAGE ERROR OF THE BEST FINGER IN THE ORDINARY TEST TAKEN IMMEDIATELY BEFORE THE "UNKNOWN FINGER" TEST; AND BELOW THAT AGAIN IS GIVEN AVERAGE OF THE "AVERAGE ERRORS PER CENT." OF ALL FOUR RIGHT FINGERS (PALMAR ASPECTS) IN THAT ORDINARY TEST.

	Day of Experiment						
	5		6		7		Average
Spot 1	22·2	..	25·0	..	14·3	=	20·5
Spot 2	42·9	..	15·4	..	33·3	=	30·5
Spot 3	56·6	..	25·0	..	66·7	=	49·4
Average of above errors	40·5	..	21·8	..	38·1	=	33·5
Average error per cent. of *best* finger in ordinary test, same day ..	41·4 (R. 2)	..	21·1 (R. 5)	..	37·2 (R. 2)	=	33·2
Average of average errors per cent. of all four fingers in ordinary test	50·9	..	37·3	..	46·8	=	45·0

A most interesting point is revealed when the average error per cent. of the three spots in each of the "unknown finger" tests is compared with the "average error per cent." of the different fingers in the ordinary test on the same day. It might have been thought that as in this test the spots are touched in an indifferent order upon the different fingers, and that as the accuracy of localization of tactile stimuli differs upon the different fingers when these are examined separately, the error in the "unknown finger" test would be of nearly the same value as that of the average of the errors of the separate fingers in the ordinary test. This is not the case. That average is given for each day on the lowest line of Table XXVIII, and it is seen to be considerably greater than the error in the "unknown finger" test. But the average error per cent. of the best finger (that on which the error was lowest) in the ordinary test approximates very closely to the average error in

the "unknown finger" test. The average error of the best finger is given immediately above that for all the fingers in Table XXVIII. Immediately above it again is given the average error in the "unknown finger" test, and the close approximation is evident. It will be observed that, upon the whole, the average error of the best finger in the ordinary test is somewhat less than the average error in the "unknown finger" test.

This result is of course obtained from a relatively small number of observations, and the smallness of these may lead to inaccuracy. But the result is a most curious one. It would seem as if in a manner the "oneness," "twoness," or "threeness" of a spot (that is, its relative position on the finger; the touches, of course, being each single) is a general character common to the spots on the different fingers if no restriction of the touched spots to a single finger is made.

But it must be remembered that in the ordinary test considerable variations occur in the accuracy of the localization of tactile stimuli upon the different fingers. This observation would seem to be antagonistic to that described above. Were the error of localization merely a negative one, and were it due to the elimination of a part of the normal mechanism or to the destruction of a part of the normal process, it would be a matter of difficulty to reconcile the two observations. But perhaps the conception of "positive error" or the distortion of the mechanism (or process) makes such a reconciliation possible.

Thus, if the condition was merely one of negative error the restriction of the test to a single finger would in no manner inconvenience the subject. If he has, as it were, a remnant of the normal process of the cerebrum whereby (on one side) the localization of tactile stimuli is conditioned, the restriction of the investigation to a single finger will in no way impede his correct localizations; while the incorrect ones will be as easily localized upon that finger as upon any other. But if there is "positive error," if the cerebral mechanism is definitely distorted in some manner or other, the subject may feel that a tactile stimulus upon the finger to which the experiment is restricted is really and definitely localized upon another finger—although he knows from the conditions of the experiment that this is not so. Some sort of definite effort is then required on the part of the subject to make the localization on the finger to which the experiment is restricted; and such efforts may well disturb the accuracy of his results. Thus spot "3" on the little finger is touched; the subject may feel that the spot touched is spot "3" on the palmar aspect of the fourth right digit; yet he knows

that it must be located somewhere upon the little finger. He then supposes that the spot is more like spot "2" on the little finger than spot "3" on that finger. The result is that he makes an inaccurate localization—whereas the place of the spot would have been correct if he had made the localization upon the fourth finger—although then the finger element in the localization would have been incorrect.

This supposition is not a fanciful one. The subject of these experiments occasionally told us—in the earlier records—that he felt that the spots which were being touched lay upon another finger than that upon which he knew (from the conditions of the experiment) they must lie. He said, "The other finger seems to draw them away."

The training was not directed to the correction of error as regards the finger upon which the spots lay, but only to the correction of error as regards the place of the spots on the finger. Distortion of the "finger" element in the "positions" of the tactile stimuli may well have been a factor in the error of localization when the test was done on restricted spots on the restricted fingers. When the restriction of the fingers was removed in the "unknown finger" test this distortion may have been removed, and the localization of the place of the spot irrespective of the localization of the finger upon which the spot lay may have been freed.

Another point must, however, be noted in connection with these observations before the training of the ordinary spots on the right index finger. Although the average error of localization of the three spots irrespective of the fingers upon which they lay in the "unknown finger" test was nearly similar to the average error per cent. of the best finger in the ordinary test (in which each finger was examined separately), the incidence of error as between the three individual spots was not similar in the two tests. Thus the localization of spot "1" might be relatively bad in the "unknown finger" test—as compared with that in the ordinary test—and so on, yet the two averages for all three spots might show close agreement.

When the errors of localization of the fingers (irrespective of localization of the spots upon the fingers) are examined it is seen that considerable variation occurs (Table XXIX).

Here the error of hazard should be 75 per cent. The table shows that this is greatly exceeded in the case of the fifth digit—the worst of the four as regards this error; and it is slightly exceeded in the case of the fourth. It was especially in the case of the little finger that the subject in the earlier records felt that the spots touched were on another

finger—usually the fourth. Here is confirmatory evidence of the existence of "positive error" in the localization of tactile stimuli upon a certain finger irrespective of their localization upon that finger itself. As it were, there was a distortion of the element of "finger" in the position of the stimuli applied to a certain set of spots. There was little or no confusion, only a definite reference of the spots to another finger.

TABLE XXIX.—THE PERCENTAGE ERROR OF LOCALIZATION OF THE INDIVIDUAL FINGERS IN THE "UNKNOWN FINGER" TEST ON THE SAME THREE DAYS AS IN TABLE XXVIII.

	Day of Experiment			
Finger	5	6	7	Average
R 2 ..	**16·7** ..	**83·3** ..	**57·1**	= **52·4**
R 3 ..	50·0 ..	28·6 ..	20·0	= 32·9
R 4 ..	66·7 ..	83·3 ..	80·0	= 76·7
R 5 ..	85·7 ..	100·0 ..	62·5	= 82·7

Tables XXX and XXXI compare respectively with Tables XXVIII and XXIX, and they give the corresponding figures for the five days after the training of the palmar set of spots on the right index finger.

TABLE XXX.—THIS TABLE IS SIMILAR TO THAT GIVEN IN XXVIII, BUT THE FIGURES NOW ARE THOSE OBTAINED ON THE FIVE DAYS WHICH IMMEDIATELY FOLLOWED THE TRAINING OF THE RIGHT INDEX FINGER. THE FIGURES IN SQUARE BRACKETS GIVE THE AVERAGES AFTER EXCLUDING RECORD "10."

	Day of Experiment.					
	9	10*	11	12	13	Average
Spot 1	0·0 ..	85·7 ..	0·0 ..	0·0 ..	23·7	= 21·9 [5·9]
Spot 2	20·0 ..	50·0 ..	20·0 ..	20·0 ..	25·7	= 27·1 [21·4]
Spot 3	37·5 ..	75·0 ..	0·0 ..	12·5 ..	50·7	= 35·1 [25·2]
Average of above errors ..	19·2 ..	70·2 ..	6·7 ..	10·8 ..	33·4	= 28·1 [17·5]
Average error per cent. of *best* finger in ordinary test, same day	16·7 (R. 2) ..	6·1 (R. 3) ..	15·7 (R. 2) ..	7·6 (R. 2) ..	23·5 (R. 2)	= 13·9 [15·9]
Average of average errors per cent. of all four fingers in ordinary test	37·6 ..	21·3 ..	31·9 ..	28·3 ..	41·9	= 32·2 [34·9]

* On this day the subject had been for too long a walk and had just come in, rather breathless, before the test.

In these tables we are, perhaps, justified in eliminating the record for the tenth day—as on that occasion the subject had just returned from a longer walk than he had yet attempted, and had hurried in the last part of it, as he was late. From Table XXX it will be seen, even including this record, that the error as regards the position of the spots irrespective of the finger upon which they were localized is in every case considerably less than before. As compared with the average of

the percentage errors of localization of each individual spot on all the fingers in the five ordinary tests after the training, the errors for the individual spots in the "unknown finger" test show considerable diminution except in the case of spot "1." Thus: ordinary test (averages obtained from Table XV)—spot "1" = 17·3 per cent.; spot "2" = 35·1 per cent.; spot "3" = 43·3 per cent.; average of the three = 31·9 per cent.; "unknown finger" test—spot "1" = 21·9 per cent.; spot "2" = 27·1 per cent.; spot "3" = 35·1 per cent.; average of the three = 28·1 per cent.

When the averages for the three spots in each record of the "unknown finger" test are compared with the averages for all three spots on each finger and on all the fingers together in the ordinary test (in which the stimuli were restricted at any one time to a single finger) it is again seen that the average in the "unknown finger" test is considerably less than the average for all the fingers in the ordinary test. [Except in the case of record "10," which we may perhaps eliminate.] On the other hand, the average error of localization in the "unknown finger" test is more near to that of the best finger in the ordinary test. Usually it is somewhat greater than the average of the best finger in the ordinary test. Except upon one occasion (record "10"—which we may eliminate) the finger upon which tactile stimuli are most accurately localized is now the trained finger—the palmar aspect of the right index finger.

It would look as if the effect of the training of the right index finger extended to the localization of the spots—irrespective of the finger upon which they are localized—in the unrestricted "unknown finger" test. Table XXXII brings out this point. In that table there is first given the average for each individual spot in the five ordinary test records, and the average of the three averages; alongside these are given the corresponding averages for these spots in the "unknown finger" test—no account being taken of the correctness or incorrectness of the localization of the fingers. The extremely close approximation of the two sets of figures will be observed. On the whole those for the "unknown finger" test are slightly greater—that is, the errors of localization are slightly greater—than for the right index finger in the ordinary test, in which the stimuli were then restricted to that finger. In this case there was at times a fairly close approximation between the individual percentage errors of the three spots on the right index finger in the ordinary tests on the separate days and the errors for three spots (irrespective of finger) in the "unknown finger" tests on the same days.

This was seen, for instance, in record "13," where 200 observations were made, about 67 stimuli being applied to each of the three spots (irrespective of the fingers—i.e., about 17 to each individual spot on each finger. On that day the errors per cent. for the individual spots on the right index finger in the ordinary test were: Spot "1" = 0·0 per cent.; spot "2" = 26·6 per cent.; spot "3" = 43·9 per cent.; average of the three = 23·5 per cent. Whereas the errors in the "unknown finger" test were: Spot "1" = 23·7 per cent.; spot "2" = 25·7 per cent.; spot "3" = 50·7 per cent.; average for the three = 33·4 per cent.

Now the inference from this would seem again to be that the effect of training the "place" element in the localization of tactile stimuli upon the right index finger was extended to the corresponding spots in the unrestricted "unknown finger" tests. Is the "finger" element in that localization also improved by the training?

TABLE XXXI.—This Table is similar to Table XXIX, but the Figures now are those obtained on the Five Days which immediately followed the Training of the Right Index Finger.

Finger	Day of Experiment 9	10	11	12	13	Average
R 2	**42·9**	**85·7**	**71·4**	**80·0**	**83·7**	= **72·7**
R 3	40·0	40·0	60·0	33·3	44·4	= 43·5
R 4	80·0	80·0	40·0	57·1	73·5	= 66·1
R 5	50·0	75·0	50·0	71·4	65·4	= 62·4

Apparently not. In Table XXXI the figures for the errors of localization of the different fingers (irrespective of the place of the spots upon them) are given. On comparison with Table XXIX (before training the right index finger, or rather, partly also after the commencement of that training) it will be seen that now the trained right index finger is definitely less accurately localized than before the training. It indeed is now the least accurately localized finger, and the error of localizing it is not markedly less than the error of hazard. The localization of the third digit is also worse than before the training; while the fourth and fifth digits are more accurately localized than before. The figures for record "13" are peculiarly valuable, as they are obtained from a large series of observations—200, about 50 stimuli applied to each finger. The percentage error of the localization of the trained right index finger is here definitely and markedly above the error of hazard. The error of hazard is about 75 per cent.; the error of localizing the right index finger is 83·7 per cent. This points quite distinctly to the presence of "positive error"—to a definite distortion of the "finger"

element in the localization of spots on the right index finger. And this even where the error of localizing these spots on that finger when the experiment is restricted to it has markedly fallen after the training.

TABLE XXXII.—This Compares the Average Error per cent. of Localization of Tactile Stimuli on each Individual Spot of the Trained Right Index-finger in the Five Records taken after the Training, along with the Average of the Three Averages. Beside them is placed the Average Errors of the Three Spots in Four of the Five Records of the "Unknown Finger" Test taken on the same days (Record "10" being left out). These Errors are Irrespective of the Error of Placing the Spot upon the Finger in this Test.

	Ordinary Test	"Unknown Finger" Test
	Records 9—13	Records 9, 11, 12, 13
Spot 1 ..	0·00 ..	5·9
2 ..	20·99 ..	21·4
3 ..	26·53 ..	25·2
Average ..	15·8 ..	17·5

There can, we think, be little doubt that in some manner the training of the spots on the palmar aspect of the right index finger has resulted in a general improvement in the localizing of the place of tactile stimuli applied to these spots irrespective of the finger upon which they are localized in the unrestricted "unknown finger" test; but that there is absolutely no improvement in the localizing of tactile stimuli upon the trained finger in that test. As it were, there may well be two elements in the "position" of the stimulus applied to a spot—its location upon a certain and perhaps rather indefinitely bounded portion of the body, and its more exact location within that region. These two elements seem to be independent to this extent, that one may show marked improvement in cases of this sort and after training specially directed to that improvement, while the other may exhibit no such improvement. Where the improvement of one element in "position" occurs it seems to be extended to other similar elements in other similar spots.

(7) *On the Question of the Nature of the (apparent) general Improvement of Localization of Tactile Stimuli which occurred in the untrained Fingers.*

In the preceding subsections of this part of the paper we have noticed that there was an apparent general improvement of localization of tactile stimuli on all the fingers—trained and untrained—in the five

days of the experiment which followed the first period of training. This improvement is seen when we examine the averages for each set of spots, and it might perhaps be inferred that in some way it also was conditioned by the training of the set of spots on the palmar aspect of the right index finger. But we have already pointed out that an examination of the incidences of error for each spot on each finger seems to throw doubt upon the accuracy of such an inference, for the general improvement in the untrained fingers is not conditioned by an increase in the accuracy of localization for the spots which correspond to those which exhibit the greatest improvement upon the trained finger.

Another possibility is that this general improvement on the untrained fingers may be due to practice; while a third (perhaps associated with the second) is that the apparent general improvement is conditioned by the disappearance of the "confusion" error.

In the earlier records of this series the subject repeatedly indicated that he was unable to make a localization, although he was aware that a tactile stimulus had been applied. It happened that, after the period of training of the palmar set of spots on the right index finger, this error practically disappeared from the records. When it occurred we counted it as a wrong localization; but it is clear that, if the subject had made an attempt to localize these stimuli, a certain proportion of them might have been correctly localized. This would have been the case even if the subject had guessed these localizations, for then one-third of them on an average would have fallen upon the correct spot.

But if a certain proportion of a number of answers which in one period of the experiment is counted as definitely wrong, is in a later period of the experiment to be counted as definitely correct, it is clear that an apparent increase in the accuracy of localization may occur. Is the disappearance of the "confusion" error the condition of the apparent general improvement of localization in the untrained fingers?

It is not easy to decide how to treat this phenomenon of the disappearance of confusion. Thus the subject was unable to localize a certain proportion of the stimuli before the period of training. But after that period he was apparently able to localize nearly all the stimuli which were applied to him. We may say that he apparently became able to localize that proportion of stimuli which previously he could not localize.

But *how* did he localize them? There are here many possibilities. Thus he may have localized this proportion of stimuli purely by hazard,

or he may have localized it all on a certain specific spot (that spot being the same one on each finger, or varying on the different fingers); or he may have localized it always on the correct spot (for the "confusion" error occurred when the stimulus was applied to any of the three spots on a finger; or he may have localized it always on incorrect spots; and so on. Finally, he may have localized this proportion of stimuli in the same ratios as between the definitely correctly localized stimuli and the definitely incorrectly localized stimuli at the time when the confusion error was present.

We may, perhaps, regard the "confusion" answer as in some manner conditioned by a stimulus the localization factor of which is subliminal; and the disappearance of the confusion error as conditioned by a fall in the localization threshold. If we accept such a view we would be predisposed to think that the previously "confused" proportion of answers would be localized in the same proportions as those which previously were definitely localized as between right and wrong. The cerebral mechanism is in some manner disturbed by the lesion, and when a stimulus can be localized certain definite errors occur. It would be expected that these same errors would be seen in the proportion of "confused" answers if they became definitely localized ones.

An interesting result is obtained if we analyse the figures for the localizations in each set of spots before and after the period of training, and endeavour to correct for the disappearance of the "confusion" error (Table XXXIII). Thus in the records before the training of the palmar set of spots of the right index finger there was: (1) a certain proportion of indefinite localizations (that is, the "confusion" answers); (2) a certain proportion of definite localizations which were correct, and (3) a certain proportion of definite localizations which were wrong. After the period of training "1" practically disappeared. Now if we take "1" and divide it in the proportions of "2" and "3" (that is, in the ratio of the correct to the incorrect answers) and take that proportion which corresponds to "3" and add it to "3," we obtain a correction of the confusion error and the resultant will be the proportion of definite wrong localizations which would be expected to be made if there was no "confusion." This has been done in Table XXXIII, and it will be seen that the computed figure for definite incorrect localization agrees very closely with the actual figure for incorrect localizations obtained in the records after the period of training and when the "confusion" answer had disappeared. But it does so

only in the case of the untrained fingers. The actual figure in the case of the trained right index finger is very much smaller than the computed.

TABLE XXXIII.—Table showing the possible Effect of Disappearance of the "Confusion" Error. Except those in Column "G," the Figures refer to the Experiments before the Training of the Right Index Finger. They are given for each Aspect of each Finger, and for the Three Individual Spots of the Palmar Set on the Right Index Finger. Column "A" gives the Percentage of Correct Localizations; Column "B," the Percentage of "Confusion" Answers; Column "C," the Percentage of Definitely Wrong Localizations; Column "D" gives the Percentage of Definite Localizations (Right and Wrong); Column "E" gives the Percentage of "Confused" Answers which would have been expected to be Wrong if the Proportion of Answers in Column "B" had been divided as between Definitely Right and Definitely Wrong in the same proportions as those in Columns "A" and "C"; in Column "F" the Figures are obtained by adding those in Columns "C" and "E" together—thus giving the Total Percentage of Definitely Incorrect Localizations if the "Confusion" Answers were Eliminated; and in Column "G" the actual Figure for Percentage Error obtained after the Training of the Right Index Finger (when "Confusion" disappeared) is given for comparison.

Finger	A	B	C	D	E	F	G
Palmar **R 2**	**57·30**	**14·37**	**28·33**	**85·63**	**4·75**	**33·08**	**15·72**
R 3	68·02	7·37	24·61	92·63	1·96	26·57	29·68
R 4	53·28	10·20	36·52	89·80	4·15	40·67	43·06
R 5	56·94	7·61	25·45	92·39	2·92	38·37	40·68
Dorsal R 2	71·44	11·79	16·77	88·21	2·24	19·01	21·62
R 3	81·24	5·50	13·26	94·50	0·77	14·03	15·72
R 4	73·20	9·18	17·62	90·82	1·78	19·40	20·62
R 5	67·38	12·49	20·13	87·51	2·86	22·99	22·16
R 2 Spot "1"	67·57	8·11	24·33	91·90	2·14	26·47	0·00
"2"	37·50	25·00	37·50	75·00	12·50	50·00	20·98
"3"	65·00	10·00	25·00	90·00	2·78	27·78	26·53

This would seem to show that the apparent general improvement of localization of tactile stimuli in the case of the untrained fingers is actually referable to the disappearance of confusion—which synchronized with the period of training; but at the same time it shows definitely that the improvement of localization in the case of the trained finger is not referable to this, and it strongly emphasizes our suggestion that this was indeed conditioned by the training.

The same table gives information with regard to the three individual spots on the palmar aspect of the right index finger. Of these, spots "1" and "2" showed a very definite improvement in localization, while spot "3" showed a much smaller improvement. When the "confusion" error before the training is corrected in the manner described above, it is found that in the cases of spots "1" and "2" the computed

figure is very much greater than the figure for the definite incorrect answers after the period of training. But the computed figure for spot "3" is only slightly less than the actual one. This seems to show that the effect of the training was to increase the accuracy of localizing stimuli which were applied to spots "1" and "2," but not to increase it for stimuli applied to spot "3"—or to increase it very slightly for that spot.

If we assume that the proportion of previously "confused" answers was definitely localized by hazard after the disappearance of the confusion—instead of being definitely localized in the same ratio as the definitely localized answers (correct and incorrect)—there is again a fairly close correspondence between the computed figures and the actual figures for definitely localized answers after the disappearance of the "confusion" phenomenon. These figures are given for the percentages of definitely localized incorrect answers in Table XXXIV—where they are compared with the figures computed in the manner described before. In each case the difference between the computed and the actual figure is given.

TABLE XXXIV.—In this Table Columns "F" and "G" are the same as the correspondingly marked columns in Table XXXIII. Column "H" gives the Difference between the Two, "+" signifying that the Real Figure (i.e., that in Column "G") is greater than the computed. In Column "I" the Figure is computed on the assumption that the previously "Confused" proportion of Answers was Localized by Hazard after the Disappearance of the Confusion—that is, that Two-thirds of this Proportion of the Answers was Incorrect. Column "K" is a repetition of Column "G"; and Column "L" gives the Difference between the Numbers in Columns "I" and "K."

Finger	F	G	H	I	K	L
Palmar R 2	33·08	15·72	−17·36	37·91	15·72	−22·19
R 3	26·57	29·68	+ 3·11	29·52	29·68	+ 0·16
R 4	40·67	43·06	+ 2·39	43·32	43·06	− 0·26
R 5	38·37	40·68	+ 2·31	40·52	40·68	+ 0·16
Dorsal R 2	19·01	21·62	+ 2·61	24·63	21·62	− 3·01
R 3	14·03	15·72	+ 1·69	16·92	15·72	− 1·20
R 4	19·40	20·62	+ 1·22	23·74	20·62	− 3·12
R 5	22·99	22·16	− 0·83	28·46	22·16	− 6·30
R. 2, Spot "1"	26·47	0·00	−26·47	29·47	0·00	−29·47
"2"	50·00	20·98	−29·02	54·17	20·98	−33·19
"3"	27·78	26·53	− 1·25	31·67	26·53	− 5·14

It will be seen that the computed figure is still larger than the actual one in the case of the trained finger; as well as in the case of the two individual spots which showed the greatest improvement in that finger. In the case of the percentage errors of localization for the palmar aspects of the untrained fingers a very exact correspondence

between the computed and the actual figures occurs. But there is a less close correspondence when the figures for the dorsal aspects of the fingers are examined. There in every case the computed figure is greater than the actual one.

It would, therefore, appear that the improvement in accuracy of localization of stimuli applied to the trained palmar set of spots (or to two of these three spots when the figures are analysed more closely) cannot be ascribed to the disappearance of the "confusion" error which synchronized with the period of training. The apparent slight general improvement in localization seen in the palmar sets of spots of the three untrained fingers may be explained as due to the disappearance of the confusion error, whichever of the two manners of correcting for that error we use; but, if it be corrected for on the assumption that the previously unlocalized proportion of answers becomes localized in the same ratios (as between definitely right and definitely wrong) that obtain in the previously definitely localized answers, it would appear that instead of an apparent slight general improvement of accuracy of localization after the training there is a slight actual deterioration. In the case of the dorsal sets of spots, if the correction is made as above there would also appear to be a slight actual deterioration; but if the correction is made on the assumption that the previously unlocalized proportion of answers was localized by hazard, there would appear to be a greater actual improvement of accuracy of localization.

There is less variation in the results if the correction is made in the first manner than if it is made in the second, and we think that we are probably justified in the assumption that, at this stage in the experiment, the general improvement in accuracy of localization of the untrained sets of spots was an apparent one only, and conditioned by the disappearance of the "confusion" error.

VI.—Clinical Description of the Case.

Family history.—J. H. L., the patient, is the eldest son in a family of three. He was born in 1886. Both parents are alive and healthy. There is no history of nervous disorder in the family.

Previous history.—The patient left school at the age of twelve, having reached Standard IV. For some years he worked on a farm with his father who was a brewer's maltster. At the age of 23 he joined the Army, and served from March, 1909, to March, 1912. On leaving the Army he worked as a platelayer on the railway.

On the outbreak of war he was called up, and went abroad with the 1st British Expeditionary Force.

With the exception of influenza in 1910 he had no illness of any description.

History of present illness.—The patient was wounded near Ypres on October 26, 1914, about 2.30 p.m. His company was in the trenches repelling a German attack. He was in a leaning attitude firing as rapidly as he could. While in the act of pressing the trigger he felt a sudden shock in his head, and lost consciousness for about five minutes. When he recovered he found himself in a sitting posture. He could feel blood running over his face. He was blind and unable to understand what his comrades were saying. At the moment when he was knocked out he had been speaking, and he now found himself unable to utter any word except "Yes."

When the Royal Army Medical Corps orderlies gave him a drink of water he was given much more than he required, as he kept on repeating "Yes," "Yes." He had a vague idea that he was being captured by Germans and started to struggle. He found, however, that he was quite unable to use either limb on the right side.

He lay in the trench until nightfall and was then removed on a stretcher to the Medical Officer's dressing station. He was very weak from loss of blood.

He was conveyed by motor ambulance to Boulogne and on reaching hospital was taken straight to the operating theatre. On waking next morning he felt very weak and unable to move. His speech had not returned, but he could now understand what people were saying and could see.

During the first day in hospital he had a vivid dream of walking under the Eiffel Tower.

His speech and ability to read remained in abeyance for about a week, but long before he could speak he was able to sing songs which he knew by heart.

During the first few days of his illness he had incontinence of faeces, but no difficulty with regard to micturition. On the fourteenth day after admission the right side of his face twitched for a few seconds. One day he saw a magazine lying on the bed next to his own and for some hours he lay trying to puzzle out the title on the front page. Suddenly it came back to him, and he alarmed the ward by exclaiming "Bystander" in a loud voice. After this his vocabulary rapidly grew, but he stammered a great deal when speaking. All parts of speech came back with equal rapidity.

On December 15, 1914, he was shipped to Southampton and sent to a hospital in Wandsworth. At this time he was still paralysed on the right side. At the end of a month he was allowed to get up. He felt very weak and with assistance could only walk a few yards.

During his residence in this hospital he suffered considerably from his eyes. He had much photophobia, and felt dizzy when he looked at objects. For a fortnight he wore an eyeshade.

With regard to his paralysed limbs, recovery of power began in the lower extremity, and was much more rapid than in the upper limb. The right side of his face was also paralysed, and at first his tongue used to fall to one side, so that he bit it during sleep.

For many weeks he was quite unaware of the position of his affected limbs in bed, and had to grope for his right hand. Five weeks after admission to hospital in London he had a "seizure" affecting the right side. He had been gargling his throat with salt and water, and swallowed some the "wrong way." While in the act of coughing violently the muscles on the right side of his body began to twitch, producing irregular movements of the right upper and lower limbs. Consciousness was preserved, and in about five minutes the attack passed off.

This epileptiform seizure was followed by a change in his mental state. On the following day he developed the delusion that other patients were making disparaging remarks about him. He had several more seizures before leaving this hospital, and found that their duration was shortened if he remained quite motionless and silent.

While in bed his right lower extremity was habitually flexed at the knee. He made an effort to straighten it one day by pressing his foot against the bed-rail. He became suddenly giddy and appears to have lost consciousness, and to have remained in a stuporose state for several days. Recovery was followed by the appearance of fresh delusions. A magazine illustration of famous generals which he saw in the hospital library suggested to him that he must be Napoleon's grandson. This delusion was accompanied by auditory hallucinations. A voice repeatedly told him of his imperial rank.

His explanation of this delusion is worth recording. The date of publication of the magazine was that of his father's birthday. This occurred to him when he was looking at the photograph of the Kaiser and Napoleon, and it at once became clear to him that he must be Napoleon's grandson.

On March 1, 1915, he was transferred to the Royal Victoria Hospital, Netley. He had so far recovered that he was able to walk to the station. His memory for this period is good. He remembers giving particulars of himself and being put to bed. He became excited and repeatedly shouted "Three cheers for the Red, White and Blue." Every time he said this a "voice" echoed his words. At the end of three days he was transferred to the Military Hospital, Maghull. At the station he struggled and was put in the guard's van. He was seized with the idea that he was being electrocuted through the floor, as he had a sensation of pins and needles in his right lower limb, and involuntary movements on the same side. This delusion persisted for some weeks. Not only did he hear imaginary "voices" talking about electrocution, but a patient in the bed next his own talked a great deal about electricity. In this way he was encouraged in his false belief.

When examined on the day after admission he was found to be dull and lethargic. He expressed delusions of grandeur, and exhibited "negativism." He had difficulty in selecting words. Articulation was slow and slurring. Paresis of both extremities on the right side was noted. He showed coarse tremor, slight nystagmus, and increased deep reflexes. Ankle-clonus existed on the right side.

During the summer of 1915 his mental state underwent but little change.

His delusions persisted, and he was inclined to be aggressive. Motor power gradually returned in his affected limbs. He continued to have fits, and on three occasions lost consciousness; usually the fits were Jacksonian in type, and presented the following characteristics. A sudden sensation of vibration or electricity in the right arm, accompanied by a feeling of faintness, warned the patient to lie down. He remained conscious, could see and hear everything, but was unable to speak. Twitching of his muscles next occurred; the thumb was always first affected; then, in order, the hand, elbow, shoulder and body on the affected side. The leg was affected last, while the face always escaped. On several occasions the head was deflected to the right. Each fit was followed by transient motor weakness in the affected limbs, while articulation became more impaired for several days.

In the later months of 1915 his attacks became less frequent and his mental state improved greatly; he became tractable and lost his delusions. Auditory hallucinations persisted until the month of November.

The following notes on his condition were made during the last week of November, 1915.

Clinical examination.—The patient is a tall, well-developed man: he is right-handed. His pulse is irregular and intermittent. His urine contains no albumen and his radial arteries are not thickened. The wounds of entry and exit, which mark the course of the bullet which penetrated the patient's skull, lie in the following situations: The former lies 12 cm. posterior to the nasion and 5 cm. to the left of a line joining that point with the inion: the latter (wound of exit) measured along this sagittal line lies 29 cm. posterior to the inion, and 3 cm. to the left of the median plane. The area of each of these cranial wounds is equal to that of a sixpenny-piece. Immediately anterior to the wound of exit there is a large trephine opening in the parietal bone, in which pulsations can be observed. It has an antero-posterior length of 11 cm., and a breadth of 4 cm. Its anterior extremity lies 22 cm. posterior to the inion and 3 cm. to the left of the median plane. Pressure on the edges of this foramen causes intense local pain. He is observant and intelligent.

We take the opportunity of remarking here how much we are indebted to the patient himself for our subsequent experiments, which would hardly have been possible without his intelligent co-operation and patience. His memory and attention are good. He has no disturbance of perception. Articulation is slow and slightly impaired; he stumbles and hesitates when pronouncing certain words. He is able to read, and writes with his left hand. In carrying out simple orders he confuses the right side with the left. For example, when told to close his right eye he almost invariably closes his left. Occasionally he uses wrong words, and has difficulty in finding the word he wants. In the addition of figures he frequently makes mistakes. There is no apraxia. He states that when excited he becomes deaf in the left ear.

Special senses.—Smell, taste, and hearing are normal. The visual fields are restricted, especially that of the left eye.

Cranial nerves.—The pupils are equal and react normally to light and on

accommodation. Ophthalmoscopic examination reveals evidence of previous retinal change. The disc margins are slightly obscured and the vessels near the disc ill-defined. There is very slight motor weakness of the right side of the face; the right eye is habitually more open than the left. When he closes the right eye there is overaction of the facial muscles on the same side. The corneal reflexes are equal. The functions of other cranial nerves are normal.

Motor system.—The left limbs are unaffected. The size of the muscles is equal in the two upper limbs, but their tone is much increased on the right side. There is slight contracture of the fingers. The range of movement is normal at the right shoulder, elbow and wrist joints, but movements of the thumb and individual fingers are much restricted. The right upper limb is much weaker than the left; the grasp of the right hand, measured with the dynamometer, is only 23 kgm., while on the normal side it is 45 kgm. Voluntary power is especially weak in the interossei and in the intrinsic muscles of the thumb. Movements of extension of the digits are much weaker than those of flexion. Adduction of the thumb is very imperfectly performed, and a movement of flexion of the thumb accompanies flexion of any of the fingers. The thumb is usually flexed at the metacarpophalangeal joint, adducted, and extended at the interphalangeal joint. Loss of voluntary movement is more complete in the fifth digit than in the index finger. Full extension or flexion of the little finger is impossible, and the attempt is accompanied by movements of other fingers. Owing partly to this motor weakness and partly to the co-existent ataxia, the limb is only used for general movements at the large joints, and is useless for finer actions. The patient holds his pipe, writes, dresses himself, &c., with the left limb.

The right upper limb is often colder than the left and slightly cyanosed.

Abrupt irregular involuntary movements may frequently be observed in the thumb and fingers. These take the form of a sudden flexion at the basal joints of one or more fingers, or an isolated movement of the thumb. They are sometimes entirely absent for several days, and are most noticeable before seizures.

The patient is unaware of their occurrence unless his attention is specially directed to his affected limb. The patient has himself observed that these involuntary movements can be increased by making strong volitional movements of the normal limbs or of the right foot; he has further observed that he can restrain them by concentrating his attention on his hand. Thus, for example, strong voluntary movements of the toes of either foot may produce involuntary movements of the affected fingers, and if, while continuing these voluntary movements, the patient directs his attention to his hand, he may succeed in arresting or diminishing the involuntary movements of the fingers. The act of yawning is accompanied by flexion of the thumb and fingers and of the wrist of the right upper limb. All movements of the right upper extremity are very ataxic. When the patient extends both upper limbs horizontally in front of him, the right arm performs swaying movements as soon as the eyes are closed.

There is no wasting of the right lower limb; the tone of its muscles is increased. There are no contractures and the range of movement is unrestricted. There is slight motor weakness in the affected limb. There are no involuntary movements. Marked ataxia exists and is well seen when the patient performs the heel to knee test.

Gait: In walking the right upper arm is adducted and remains rigidly applied to the side; movement takes place at the elbow, the forearm swinging loosely. The right lower limb executes a slight movement of circumduction at the hip. In descending or ascending stairs it is raised higher than necessary, and is then planted in an uncertain manner. The right shoulder droops perceptibly.

Reflexes.—The right arm-jerks are exaggerated; the knee- and ankle-jerks are exaggerated. Those on the right side are brisker than on the left. Ankle-clonus cannot be obtained. The plantar reflex is usually absent on the right side, on the left a normal flexor response is obtained. After a Jackonian fit, however, an extensor response exists for some hours on the right side. The abdominal reflexes are equal on the two sides.

Subjective sensations.—He has frequent headaches on the left side of the head, and local pain in the neighbourhood of the cranial wound. In his right upper limb he has often a feeling of numbness, and sometimes there is a feeling as though the whole of the right side did not exist. The knowledge of position of his right upper limb is very imperfect when he is without the guidance of vision.

Tactile sensibility.—He cannot appreciate cotton wool touches with certainty on the tips of the fingers of the right hand, nor on their dorsal surfaces. On these areas his answers are irregular, and he often entirely fails to appreciate the stimulus. Elsewhere on the right upper limb cotton wool touches are recognized, but there is a subjective difference: the feeling is "weaker" on the right side and "not so tickly" as on the normal side. The same difference is felt on the right malar region, and on the right ear there is a "vast difference." On the right side of the abdomen and on the right lower extremity, a wisp of cotton wool rubbed across the skin seems less distinct than on the left side. He is unable to define exactly this feeling, and states that on hair-clad parts the cotton wool produces a "creeping sensation," which is diffuse, being felt over a wide area of the leg. For the purpose of examining with graduated tactile stimuli, hairs mounted on matches were roughly calibrated by measuring on a balance (in grains)[1] the force exerted by them in bending. A series of nine hairs exerting pressures varying from $\frac{1}{2}$ to 140 gr. was obtained. The method of applying the graduated tactile stimuli was that adopted by Head and Holmes. A hair is selected producing a stimulus just above the minimum threshold to which the normal hand reacts with constancy when the stimulus is applied sixteen times in the minute: the same hair is used in the same manner to a corresponding part of the affected hand, and then more powerful hairs are used, up to those exerting many times the pressure.

[1] Metric system weights were unobtainable in this hospital.

The strength of the stimulus is then decreased to the hair with which the testing was begun, ending up with a series of contacts on the normal hand.

The following result was obtained when hairs of various bending strains were applied sixteen times in the minute to an area of skin on the dorsal aspect of the hands (between the third and fourth metacarpal bones).

A stroke represents a correct answer; a nought, that the patient did not respond; a broken stroke represents an hallucination.

Hairs (bending strain in grains)	Normal hand (left)	Affected hand (right)
7	1111111111111111	—
7	—	0110000000100000
12	—	0001011111110111
20	—	1111111111111010
30	—	1111111110111011
40	—	1111111110011011
80	—	1111011111111111
40	—	0111111010111100
30	—	1111111011111100
20	—	0001111100100011
12	—	0000100111010010
7	—	0100001010000000
7	1111111111111111	—

Thus it is apparent that on the normal hand a perfect series of answers can be obtained with a hair of 7 gr. bending strain, but on the affected hand the answers are slower (the patient remarking, "It don't send the message to the brain quite so soon") and inconstant. There is a persistence of sensation, and hallucinations occur. No definite threshold is obtained. On the flexor surface of the right forearm the answers are still irregular, and are much disturbed by hallucinations and persistence of sensation. On the upper arm (bicipital area) evidence of a definite threshold is obtained, and there are no hallucinations. On the face a threshold can be obtained with hair "3." The record is not disturbed by hallucinations, and there is no slowness in response. The sensation evoked by the hair is not quite similar on the two sides. On the sole of the foot it is possible to obtain evidence of a definite threshold; hallucinations, however, are numerous. On the dorsal surface of the foot, on the inner aspect of the leg, and on the right side of the abdomen (umbilical level) the answers continue to be slow, irregular, and disturbed by hallucinations.

The following record illustrates the condition.

Abdomen (Umbilical Level).

Hairs	Normal side (left)	Affected side (right)
3	1111111111111111	—
3	—	1111111111111111
8	—	1111011111111111
30	—	1111111111111111
40	—	1111111101111111
70	—	1111011111111111
90	—	1111111111101111
70	—	1111111111110001
40	—	1101110011111111
30	—	1101111101111111
8	—	1110010100101111
3	—	1001110110010100
3	1111111111111111	—

Pressure touch.—Tested roughly with the pulp of the observer's finger or by a blunt-pointed rod (whose movement through a cylinder is resisted by a rubber band), showed an uncertainty of response, and a tendency to evoke hallucinations on the right hand and forearm. Pressure touch on the right side of the body does not always evoke the same sensation as on the left. Thus, pressure with the blunt end of a pencil on the right cheek, or on the right side of the abdomen, evokes a sensation as though a much broader object were being applied. There is a feeling of "spread-outness" which is not experienced on the normal side.

Roughness.—The same thresholds are obtained on both sides with Graham Brown's æsthesiometer. With sand-paper, four grades of roughness can be readily identified. The relative roughness of any two grades can also be distinguished. No difference can be detected between the two sides.

Tickling and scraping.—No difference to tickling or scraping can be detected between the palms or the soles of the feet.

Sensibility to pain. (*a*) *Superficial pain.*—When tested with a needle on the right cheek and on the right forearm, the patient states that the prick is less sharp than on the normal side, but the pain evoked is as great. On the right upper arm there is slight hyperæsthesia to pinprick. On other areas no difference can be detected.

(*b*) *Pressure pain.*—No difference between the two sides can be obtained. Measured prick was not employed.

Vibration.—On the right side the vibrations of a heavy tuning-fork (C) evoke sensations which are "not so plain" as on the normal side. The vibrations are appreciated for a shorter period on the right upper limb than on the left. Thus, when the patient can no longer appreciate the vibration on the right radial styloid process, it can be recognized for a period of seventeen seconds on the corresponding point on the left side. After the tuning-fork can no longer be appreciated on the left styloid process it is recognizable on the right side for a period of twelve seconds. There is thus a difference of five seconds between the two sides, and on the back of the hand it is much greater. No difference between the two sides can be obtained on the upper arm or on the lower limb.

Temperature.—The paretic limb is frequently colder than the left, but when this condition is not noticeable, definite changes in the response to thermal stimuli can be demonstrated. Thus, on the normal left palm the neutral zone lies between 27° C. and 31° C. On the right palm all temperatures between 26° C. and 37° C. appear to be neither hot nor cold. The power of comparing thermal stimuli is also defective. On the right side temperatures of 41° C. and 47° C. are correctly appreciated as hot, but he is quite unable to say which is the hotter. Similarly, he cannot discriminate between water at 15° C. and water at 20° C., whereas on the normal side he can always tell which of the two is the colder. Further, his answers on the right side are slow and irregular. On the sole of the right foot the neutral zone lies between 23° C. and 25° C.; on the normal foot it lies between 28° C. and 30° C. Here

again, however, his ability to discriminate the relative warmth of two tubes is diminished.

The application of a heat stimulus sometimes evokes a burning or slightly painful sensation on the right foot, when on the left side it is quite comfortable. On the same area a cold stimulus is described as feeling "all over the place," whereas on the normal side it is localized to one area. He has noticed while washing that water which is only sufficiently hot to be quite comfortable on the normal hand may cause a "stinging pain" on the right hand, which persists for several minutes after withdrawal.

Sense of position. (*a*) *Recognition of posture.*—The patient's recognition of the posture of his right upper limb is grossly defective. If the defect is measured by the method introduced by Horsley, and modified by Head and Holmes, the records obtained when the affected hand seeks the normal forefinger are very much better than when the patient attempts to point to the affected forefinger with his normal hand.

The examination of separate segments of the limb (by asking the patient to imitate, as exactly as possible with his normal limb, the posture of the affected part) shows that a grave loss is present, which is most profound in the distal segments of the limb. When one of the fingers or the thumb is placed in a particular attitude while the patient's attention is diverted, he is quite unable a moment later to imitate successfully the posture with his normal digits.

With regard to movements of the hand, at the wrist-joint he is less inaccurate, but confuses an attitude of flexion with one of extension. He does not imitate with the normal forearm attitudes of supination and pronation. The upper arm also shares in the defect, but in a much less marked degree. Recognition of posture is also impaired in the lower limb.

(*b*) *Rocognition of passive movement.*—The appreciation of passive movements is very much impaired. Movements are appreciated long before their direction can be recognized. Indeed, he is quite unable to recognize the direction of movement in the thumb or in any finger, unless hyperextension or very flull flexion is carried out. All the digits can be moved through a wide angle before the movement is perceived. At the wrist extreme flexion or extension only can be recognized, and movements through a very wide angle cannot be appreciated. Recognition of the direction of the movements of pronation and supination is grossly defective.

At the elbow-joint the direction of movement is usually correctly appreciated, when the angle of movement exceeds 60°.

At the shoulder-joint these defects are much less obvious. The recognition of passive movement and the direction of movement is also very defective in the toes and ankle. The occurrence of numerous hallucinations of movement can be noted at all limb joints, except those of the shoulder and elbow.

Localization, as tested by the modified Henri method, is found to be much affected on the right hand and forearm; and, to a lesser degree, on the right upper arm, right side of the face, and right lower extremity. Occasionally (on the right side) the patient states that he has no knowledge of the locality

of the contact, but in the majority of instances he localizes the stimulus in some false direction. This holds good, not only for the proximal segments of the limb, but also for the right fingers and palm.

For testing the patient's power of recognizing the position of a spot stimulated the pressure-touch instrument was used, the touches being repeated until the patient could appreciate the contact.

The following record was obtained by making the contacts in an irregular order on the dorsal aspect of each digit:—

Digit Normal hand (left)	Correct	Localized proximal	Distal	Localized on wrong digit	"Don't know"
Thumb	10	0	0	0	0
Index finger	10	0	0	0	0
Middle ,,	7	1	1	1	0
Ring ,,	8	1	1	0	0
Little ,,	7	1	2	0	0

Abnormal hand (right)	Correct (or on same level)	Proximal	Distal	Wrong digit	"Don't know"
Thumb	1	2	1	6	0
Index finger	2	2	0	6	0
Middle ,,	2	3	0	5	0
Ring ,,	2	1	0	7	0
Little ,,	2	0	1	7	0

Thus on the normal (left) digits he correctly localizes 42 contacts out of 50; while on the abnormal (left) digits only 9 are correctly placed. On another occasion, out of 20 painful stimuli equally distributed on the terminal phalanges of the right digits only one was correctly localized, 5 were incorrectly localized on the base of the thumb, and in three cases the patient had no idea of the situation of the stimulus. The dorsum of the hand was selected for a comparison of the accuracy of localization of tactile stimuli with accuracy of localization of painful stimuli; 35 touch stimuli and (afterwards) 35 pain stimuli (pin prick) were given; 7 stimuli in each case being applied to the area of skin above each metacarpal bone. The stimuli were applied in an irregular order and the patient attempted to localize them in the usual manner upon the hand of a model.

In the following table the distances of the points wrongly indicated from the spots touched or pricked are recorded in centimetres (to the nearest whole centimetre)—"C" indicating that the stimulus is correctly localized:—

Skin areas over	i	ii	iii	iv	v	vi	vii (stimuli
Tactile stimuli (dorsum of right hand).							
1st metacarpal	10	8	4	8	6	10	8
2nd ,,	6	6	3	2	5	C	2
3rd ,,	C	5	4	2	5	3	5
4th ,,	1	1	1	4	C	5	2
5th ,,	4	C	C	1	4	2	2
Painful stimuli (dorsum of right hand).							
1st	1	3	2	2	1	6	1
2nd ,,	2	2	1	2	2	2	C
3rd ,,	2	2	C	6	C	2	C
4th ,,	2	1	1	C	1	C	1
5th ,,	1	3	2	3	C	3	2

On the normal (left) hand in only one instance did the error in localization exceed 2 cm.

It would appear from the above observations that erroneous localization is present, both where the stimulus is one of pressure-touch and where it is a prick, but that in the latter case the error is not so great as in the former.

On the terminal phalanges this difference is less obvious. Out of 50 touch stimuli 12 are correctly placed, while out of a similar number of prick stimuli 17 are correctly localized (in 11 instances he had no idea of the locality of the stimulus). The patient remarked, "A prick seems often to concentrate at the wrist."

On the right forearm the power of localization is plainly defective, but the defect is not so great as in the case of the hand of fingers. Out of 20 pressure-touch stimuli 10 are localized in false positions which vary from 5 to 14 cm. in distance from the spot touched. In no instance does the patient fail to make a localization.

On the right upper arm localization is also disturbed, but is more accurate than on the right forearm.

Comparison of the accuracy of localization on the two sides of the face demonstrates a slight disturbance of localization on the right cheek.

The power of recognizing the position of stimulated spots on the right foot is much impaired. Out of 50 tactile stimuli applied either to the great toe or to the little toe, the number of errors on the left side is 7 and on the right side 26. The defect is present, although in a lesser degree, in the case of the right leg.

Compass test.—The power of discriminating the two points of the compasses is gravely affected on the right upper limb.

On the palm the patient cannot recognize the "two-ness" of two points synchronously applied, even when the distance between them is 6 cm.; whereas, on the normal left palm, no error occurs when the points are 1·5 cm. apart.

On the right side no threshold can be obtained for the fingers, palm, nor for the dorsal surface of the hand; nor can a threshold be obtained for the right forearm (neither surface), right upper arm, right side of the face, right forehead. Neither can a true threshold be found for the right lower extremity, nor for the right side of the abdomen. The power to recognize the double nature of the compass points is also lost on the right hand, when the points are applied successively.

The following are some of the records obtained:—

		Normal (Left)		Abnormal (Right)	
Palm	1·5 cm.	1: 11 111 1 11 11 2: 111 1 1 11 11 1	1·5 cm.	1: 11 1111X 11X1 2: X1X 1XX XX X1	
			4 cm.	1: X XX 1XX 1X 1 1 2: 1 1 111 11 1 1X	
			6 cm.	1: 1 11 111 111 X 2: X1XX 11X 1 1X	

		Normal (Left)		Abnormal (Right)
Forearm (flexor surface)	5 cm.	1 : 111 11 1 111 1 2 : 11 1111 111 1	5 cm.	1 : 11 1 111 1111 2 : X X XX XXX XXX
	15 cm.		15 cm.	1 : 1 1X XXX X XX1 2 : X1X 1X 11 11 1
Upper arm ..	6 cm.	1 : 1 111 1 111 1 1 2 : 11 1 1 11 111 1	6 cm.	1 : 11 111 X 10 11 2 : X 11 111 XXX X
			10 cm.	1 : 111 1 X1X 1 X1 2 : 11X X11 11 11
Face (cheek)	2·5 cm.	1 : 111 11 1 11 11 2 : 1 111 11 11 11	3 cm.	1 : X11 X X XX X 1 1 2 : 1X1 1 1 1X 1X 1
			6 cm.	1 : 11 11X 11 11 1 2 : 11 11 XX1 11 1
Sole of foot	2·5 cm.	1 : 1 1 111 111 11 2 : 11 11 111 1 11	4 cm.	1 : X 1X XX 1XX X 1 2 : 1 11X XX 1 X1 X
			10 cm.	1 : 1 11 11X 11 11 2 : X1X 11 1X X 1 1
Dorsum of foot	4 cm.	1 : 11 1 11 111 11 2 : 11 111 111 1 1	5 cm.	1 : XX 1 1X1 XXX 1 2 : X111 1 1 XX 11
			10 cm.	1 : 1 11X 1X 1X1 1 2 : 111 11 1 11 XX
Leg (inner aspect)	6 cm.	1 : 1 111 11 11 11 2 : 11 1 11 111 1 1	8 cm.	1 : 1 11 111 1 11 1 2 : XX1 X XX X X XX
			15 cm.	1 : 111 X 1 111 11 2 : 1 X1 11 XX X XX
Pectoral region (3rd rib)	7 cm.	1 : 1 11 111 1 11 1 2 : 1 11 111 11 1 1	7 cm.	1 : 111 11 1 X 11X 2 : 1 11X X1 11 11
			15 cm.	1 : X 1XX 11 1 111 2 : 111 1 1 11 11 1

His replies are characterized by slowness, uncertainty, and irregularity. On some occasions, when twoness is recognized, he states that the points feel very close together—although actually they may be separated from one another by as much as 5 cm.

Power of recognizing weight.—On the normal hand his power of appreciating weight is quite acute, but on the right hand it is much impaired. With the hand completely supported he can easily distinguish, on the left (normal) side, between 4 oz. and 8 oz., and 16 oz. and 24 oz., but on the abnormal right hand he cannot.

The power of recognizing addition and subtraction of weight is also much impaired.

With unsupported hands, when a weight is placed in each hand and the patient is asked to " weigh " them by raising and lowering his hands, the errors are equally gross. He thus cannot distinguish with certainty between 1 lb. and $1\frac{1}{2}$ lb.; nor between 1 lb. and 2 lb.

Appreciation of size.—The patient has completely lost the ability to recognize differences in size of objects placed upon his right palm, nor can he distingush between the head and the point of a pin when they are gently applied to the skin.

Appreciation of two-dimensional shape.—He is equally at fault when asked to recognize various shapes when they are placed in contact with the surface of the right palm; on the left normal palm he has no difficulty in distinguishing them.

Appreciation of three-dimensional form.—The patient is quite unable to recognize common objects placed in his right hand when his eyes are closed. These objects are at once recognized when placed in the left hand. Thus:—

Object	Reply—right hand	Reply—left hand
1. An ink-pot	"Something hard, solid and cold."	"An ink-pot" (immediately).
2. A large tuning-fork	"Something hard, light and cold."	"A tuning-fork."
3. A rolled-up handkerchief	"Something soft and tepid."	"A handkerchief."
4. Small nail-scissors	"Cannot describe it."	"Nail-scissors."
5. A penny	"Just a feeling in my hand."	"A penny."
6. A large pocket-knife	"Hard, that's all I can say."	"A knife."

Appreciation of texture.—In spite of his power of recognizing roughness or smoothness, he has great difficulty in appreciating texture with the right hand. The following record illustrates his condition:—

Material	Reply—right hand	Reply—left hand
1. Velvet	"Soft, like rubber."	"Velvet."
2. Silk	"Coarse calico."	"Silk."
3. Towel	"Ribbed surface"	"Coarse towelling."
4. Flannelette	"Like calico."	"Flannelette."
5. Satin	"Not quite satin."	"Satin."
6. Velvet (again)	"Piece of coarse substance."	"Velvet."

With his right hand he is quite unable to appreciate any difference between velvet and flannelette; with his left hand he can distinguish them without difficulty.

BIBLIOGRAPHY.

[1] Head and Holmes. *Brain*, 1911, vol. xxxiv, p. 102.
[2] Russell and Horsley. *Brain*, 1906, vol. xxix, p. 137.

RECORDS OF SPEECH IN DISSEMINATED SCLEROSIS.

BY E. W. SCRIPTURE, M.D., PH.D.

§ 1.—Method of Recording and Analysis.

Speech in disseminated sclerosis has been described in so many conflicting and indefinite ways that an attempt at accurate fixation by graphic records and analyses of the curves seems desirable.

The speech is recorded by the phonautograph method. The patient speaks into the mouthpiece of a wide rubber tube (fig. 1). This leads to a metal tube covered by a flexible membrane (fig. 2). The movements of the membrane are recorded by a lever writing on a moving blackened cylinder.

A record of "ah" sung by a normal voice is reproduced in fig. 3. The waves begin faintly; they rise quickly to full intensity. They are quite regular. In the record of a vowel each wave represents one vibration of the glottis. The length of a wave records the duration of a vibration. Slow vibrations, which produce low tones, are recorded as long waves; fast vibrations, or high tones, as short waves. Since the speed of the recording drum is known, a measurement of the length of a wave will give the duration of the vibration and the pitch of the tone. For example, with a surface speed of 1 mm. equal to 0·0085 sec., a wave of 0·9 mm. is the record of a vibration whose duration was 0·0085 x 0·9 = 0·00765 sec. If one vibration occupied this time, then the frequency, or the number of vibrations per second, would be 1 ÷ 0·00765 = 131.

Measurement of the first six waves in fig. 3 gives the following results:—

Wave length	0·9 mm.	0·9 mm.	0·9½ mm.	0·9½ mm.	1·0 mm.	1·0½ mm.
Duration	0·00765 sec.	0·00765 sec.	0·00808 sec.	0·00808 sec.	0·00850 sec.	0·00893 sec.
Frequency	131 vib.	131 vib.	124 vib.	124 vib.	118 vib.	118 vib.

On cross-section paper the curve is supposed to be laid along the X-axis and a dot to be placed above each wave at a height proportionate to the frequency. Such a "melody plot" for the entire record of fig. 3 is shown in fig. 4. A line drawn smoothly through these dots shows

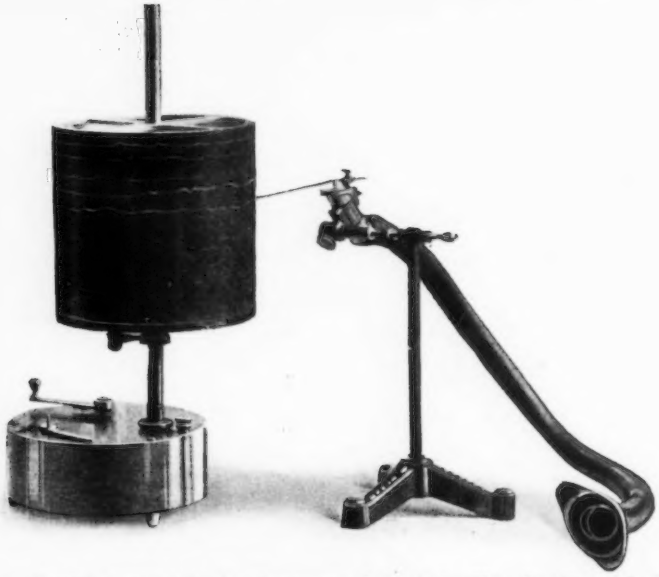


FIG. 1.—Recording speech by the phonautograph method. The vibrations and puffs of air pass to a flexible membrane, whose movements are recorded on a revolving drum by means of a light lever.

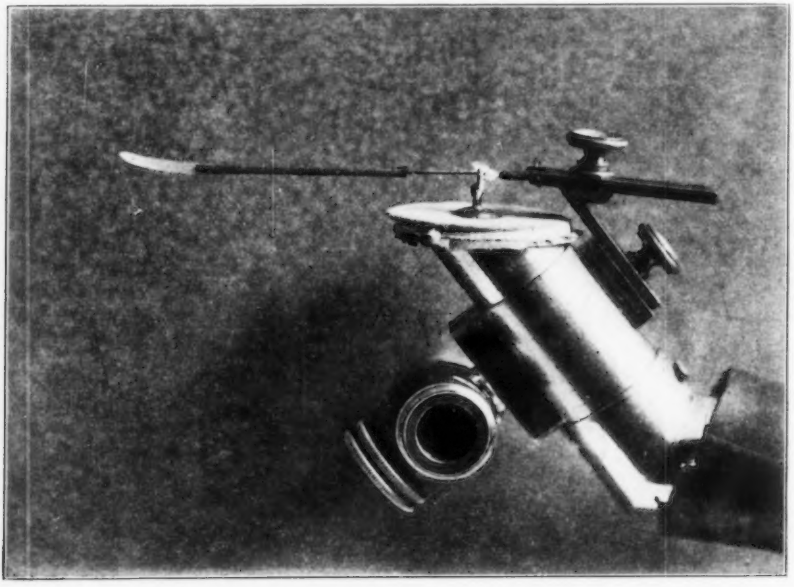


FIG. 2.—Speech recorder. The wide metal tube is covered with oiled silk, whose movements are amplified by a light lever. This recorder is a development of the Scott phonautograph by the Abbé Rousselot.

how the voice rises and falls. If the larynx could produce a tone of absolutely constant pitch, the melody plot would be a straight horizontal line. Actually the melody plot, even in song, is a perfectly straight line only for short distances.

A record of "I'd like to go home" by a normal voice is given in fig. 5. It begins with the vibrations of the larynx during "I." These gradually increase in intensity. They are suddenly cut short by the descent of the line which records the movement of the tongue in closing the mouth to make the "d." The line at the base level records the sound while the tongue was held firmly across the mouth-passage—the "occlusion" of the "d." It has faint waves; these

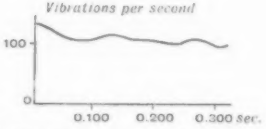


FIG. 4.—Melody plot to fig. 3. The length of each wave in fig. 3 is measured. The duration and frequency are calculated. The record in fig. 3 is supposed to be laid along the X-axis. A dot is placed above each wave on the scale of frequency. The series of dots is the "melody plot." A line drawn through them shows that the voice fell rapidly for the first few vibrations and then fluctuated with a slow fall just above 100 vibrations per second.

show that the larynx vibrated during the "d." The stronger waves that follow belong to "l" and later to "i." They are cut short by a fall not quite so sudden as for "d"; this is produced by closing the back of the tongue against the hard palate and velum to produce the "k." The straight line is ended by a sudden rise. We know that the latter portion of the straight line represents the occlusion of "t" and the sudden rise its "explosion," because in the spoken words the "k" sound is immediately followed by "t" without any break. There are no vibrations at this place, therefore the larynx was silent during "k" and "t." After the rather breathy explosion of "t" there follow a few waves that register the very short indefinite vowel "o" of "to." The line for "g" is like that for "d." The long series of large waves

FIG. 3.—Record of "ah" sung by a normal voice. Each wave registers one vibration of the larynx. The waves begin smoothly and evenly. The height rapidly increases during the first ten waves. The waves are apparently of nearly even height and length throughout. The slight general rise and fall of the record indicates some variations in breath pressure.

registers the sounds between "g" and "m." These include the two vowels "o" and the "h" between them. In normal speech the larynx usually continues to vibrate during an "h" between two vowels. The "m" is recorded by a base line with faint waves ending in a kind of explosion.

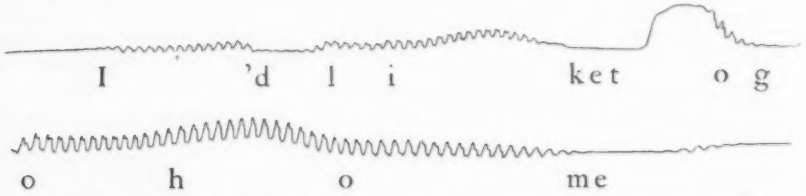


FIG. 5.—Record of "I'd like to go home," by a normal voice. The vibrations of the vowel "I" are cut short by a fall of the line at the beginning of "d." Faint vibrations are seen during the "d." The larger vibrations for "l" are followed by still larger ones for "i." These are cut short as the line descends for "k." The straight line for "k" is continued for "t"; there is no division between them. There is a strong upward movement that registers the puff of air (explosion) at the end of "t." The vowel "o" is very short. The "g" has faint waves. From the beginning of "o" through "h" and the next "o" there is an unbroken series of strong vibrations; the "h" between the two vowels is itself made into a vowel. The faint vibrations of "m" end in stronger ones.

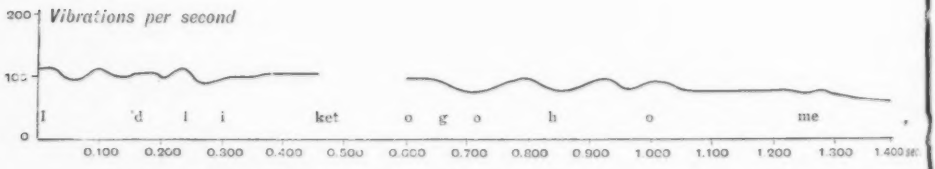


FIG. 6.—Melody plot to fig. 5. The speech curve in fig. 5 is supposed to be laid along the horizontal axis. The whole phrase lasted 1,400 seconds. Above the point where each wave of the record would come, a dot is placed that indicates on the vertical scale the number of vibrations per second that would be made by a tone with this wave length. The series of dots is the "melody plot." A line drawn through these dots indicates the rise and fall of the voice. The melody is seen to be constantly rising and falling in smooth fluctuations: this is a fundamental characteristic of a normal voice. For "kt" there are no dots, because the larynx does not vibrate during those sounds. The phrase starts at a tone of about 120 vibrations a second and falls slowly to one of about 60 at the end; the fall of an octave is the most frequent one in declarative sentences spoken by a Londoner.

The melody plot for fig. 5 is given in fig. 6. The dots lie somewhat irregularly around a line that is drawn to indicate their general position. These irregularities indicate on a magnified scale the normal minor irregularities of the laryngeal vibrations. The general course of the laryngeal tone is indicated by the line itself. We notice that the vowel starts at a little above 100 vibrations per second in "I." It fluctuates up and down within moderate limits during the sounds that follow. At "k" it suddenly stops, because the larynx does not vibrate during

this sound and the following "t." For the remainder of the sentence it continues the tone at about 100 vibrations for an instant and then gradually falls with small fluctuations to the end. These small fluctuations are essential characteristics of normal speech.

§ 2.—Records of Cases with no Perceptible Speech Abnormality; Demonstration of Laryngeal Ataxia.

The patient, E. B. E. (National Hospital), female, aged 32, stated that at 21 years of age she had felt numbness in the right leg. At the age of 28 she lost entirely the use of the right arm and leg. This was attributed to "neurasthenia" by the family physician. She became apparently well in about a year and did not notice anything troublesome. Two months ago she had noticed that the right leg was shaky and that her gait was unsteady. On examination the knee-jerks were found to be greatly increased on the right and slightly on the left. There was some uncertainty of the right hand only. Some nystagmus was present. Nothing abnormal could be detected in the voice. She herself had never noticed anything abnormal, although she sang considerably.

Records of this well-established case with no perceptible voice signs showed that the consonants were all correctly formed, and that there were no changes in intensity, duration, melody or rhythm. The vowel curves, however, revealed a peculiar abnormality. Instead of the even series of waves as in fig 3, the record of a vowel by this patient invariably showed the presence of peculiar irregular waves such as may be seen in the piece of vowel reproduced in fig. 7.

Fig. 7.—Beginning of "ah" sung by E. B. E. The waves at the start show peculiar vibrations.

The patient, E. C. (National Hospital), aged 47, had had diphtheria at 17 years of age. At 20 she suddenly lost the sight of the right eye for a time. At 27 she lost the use of the left hand temporarily; the right leg became troublesome some time later. At 37 there was entire loss of speech for a fortnight. At 45 there was difficulty in micturition. Lately the left leg was painful and the walk bad. She had had no speech trouble since the temporary one referred to above. She says she has had trouble in singing hymns ever since the attack of diphtheria. Examination showed: nystagmus and pale discs; intention tremor most marked in right extremities; very bad walk; no abdominal reflexes; exaggerated knee-jerks and ankle-clonus: extensor plantar reflex on right; diminished sensitiveness of right leg. No defects of speech could be observed by the ear except that she seemed to become tired when speaking.

A piece from a record of "ah" spoken by this patient is reproduced in fig. 8. At the start it shows the same peculiar vibrations as those seen in fig. 7 There were no other abnormalities in the speech record.

The patient, H. H. (National Hospital), had been wounded in the left hip, left arm and left shoulder during the Boer War in 1901. After being in the hospital for two months, he was invalided home. On coming out of the hospital he noticed a slight lameness in the right ankle. This became gradually worse. He became lame in both legs and seemed to "walk from the hips" (1911). Under treatment in 1912 he began to improve gradually. Some years ago he had had double vision but had recovered perfectly after three months. His arms had never been affected. There had never been any trouble in speech. On examination his walk was poor and he looked tired. The knee-jerks were both exaggerated; there was ankle-clonus on both sides. Otherwise there was nothing abnormal. His speech, in particular, seemed perfectly normal.

FIG. 8.—Beginning of "ah" spoken by E. C. The peculiar vibrations are seen at the start.

FIG. 9.—Beginning of "I'd like to go home," by H. H. After several peculiar vibrations the waves of the initial vowel become regular.

At one place only in the records does anything abnormal appear. The beginning of one registration of "I'd like to go home" is reproduced in fig. 9. The peculiar vibration is evident.

Before going further it will be well to consider the meaning of the peculiar vibrations referred to.

In the first place they have been found in the records of every case of disseminated sclerosis so far recorded (twenty in all), regardless of whether any speech defect could be detected by the ear or not. If this continues to happen as the number of cases increases, we approach nearer and nearer to a firm belief that disseminated sclerosis always reveals itself in these peculiar vibrations.

Each wave in the record is a registration of a vibration in the tone from the larynx. If the larynx could produce a tone of perfectly constant pitch, all the recorded waves would be of the same length.

When a rising or a falling tone is recorded, the waves change gradually in length, from short to long and from long to short. The peculiar vibrations do not show rising or falling tones, but are the records of very brief imperfections.

The differences and changes in pitch of the laryngeal tone are produced by altering the tension of the vocal cords. Shortening waves indicate increase of tension, lengthening waves indicate decrease of tension. The peculiar waves noticed in all the records are registrations, not of ordinary changes of tension, but of jerky irregularities of tension of the vocal cords.

The vibrating body in the larynx that produces the tone consists of the vocal muscles (usually called the vocal cords) and the thyreo-arytenoid muscles (formerly called the external thyreo-arytenoid). These are stretched by the action of the crico-thyreoid muscles. The tension—and consequently the pitch of the tone—is controlled by the interaction of these muscles. Normally the action is smooth and the waves of the vowels are regular as in fig. 3. Even the most rapid shake of the voice that can be produced voluntarily is registered as a series of even waves in which the eye cannot detect any variations. When the waves of such a tremor are measured under the microscope, the change in pitch is found to be even and never jerky. Indeed it is quite incredible that any voluntary movement can be so suddenly jerky as those recorded for disseminated sclerosis. The time occupied by the crico-thyreoid jerk in these records is often less than 0·005 sec. The fastest time yet recorded for tapping with the finger is 0·07 sec., and for a voluntary shake of the crico-thyreoid is 0·16 sec.

The involuntary shaking or quavering of the voice in emotions is not more rapid than that which can be produced voluntarily; it never consists of brief jerks. Records of real tremor of the voice such as occur in neurasthenia, senility, paralysis agitans, &c., differ from voluntary tremor only in the period of oscillations in pitch. They never show the peculiar vibrations. Both shaking and tremor of the voice consist of oscillations in pitch, not of brief jerks.

Peculiar irregular vibrations in the vowels have been found in the records of diseased conditions of two kinds. In the one class the vocal cords flap loosely instead of vibrating firmly. I have found such waves in the records of progressive bulbar paralysis, and of a normal voice with rattling crico-arytenoid joints, &c. In all these cases the irregular waves were more or less irregular throughout or for long stretches of the vowel. There was never a brief jerk at the beginning

or the end of a vowel. The other class includes the spastic conditions. Records of spastic speech occasionally show irregularities in the wave lengths at the beginning of vowels.

The discharge from a motor nucleus depends on the condition of the nucleus itself. Irregularity in the discharge indicates a defect of control. It is well established that for all muscular movements there is a system of control, termed "taxia," which co-ordinates them so that the resulting motion is properly and smoothly carried out. A defect of the control is called "ataxia." Here we have a defect in the control of the crico-thyreoid muscle in tensing the vocal cords. The peculiar vibrations are thus records of laryngeal ataxia.

The peculiar vibrations were found in these cases nearly always at the beginning or the end of a vowel, rarely during the main part of the vowel. When the adjustment of tension has been once attained, the patient seems to have little difficulty in keeping it; the ataxia appears when he tries to attain an adjustment or to let go of it. This also explains why the voice defect is unperceived in such cases, except perhaps as involving some fatigue. With more marked cases the peculiar vibrations appear at various other points.

If laryngeal ataxia is thus established as a sign of disseminated sclerosis that can be registered at a period when no speech trouble can be detected by the ear, we can hope to detect the disease at the earliest possible moment.

§ 3.—Records of Cases with Various Abnormalities; Intention Tremor; Breathing Ataxia; Staccato Speech; Elision and Attraction; Anataxia; Duration of Sounds; Scanning Speech.

Some of the further peculiarities of speech in this disease will now be studied.

J. H. (National Hospital), tramway motorman, aged 42, fell four years ago when his tram was struck. He was reported as suffering from contusion and shock. Being unable to go back to work he was examined by a neurologist. He admitted that he had noticed a difficulty in walking and speaking before the accident, but claimed that the trouble had greatly increased since then. Examination showed a well marked case of disseminated sclerosis with particular affection of the speech organs and the left leg. His claim against the tramway company for compensation was partially admitted. His speech appeared very laboured and tight. The larynx could be seen to move up and down irregularly in the neck. In a throat examination the glottis could be seen to open and shut irregularly while "ah" was sung.

When first interviewed, J. H. seemed to have some of the excessive contraction of the lips and tongue that is usual in stutterers. This disappeared shortly as he became at ease, and no expression could be obtained in the records. He admits some trouble in speech in childhood, and the nature of his speech when excited shows him to have been a stutterer.

Fig. 10 is from a record of "ah" sung by J. H. The beginning of the record shows the peculiar vibrations in a most marked manner; they occurred constantly in his records. The figure also shows that the vowel waves swell and fade away in alternations of intensity. The melody plot is given in fig. 11. During

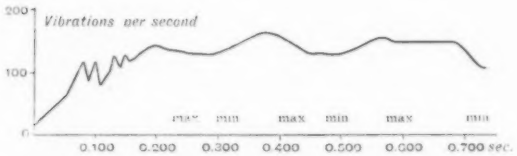


FIG. 11.—Melody plot to fig. 10. Violent jerks appear at the start. The pitch of the tone varies considerably. There is no definite relation of the pitch to the maxima and minima of intensity.

the louder portions the vowel waves themselves were shorter and during the weaker portions longer. There were thus two factors in the irregularities, namely, intensity and pitch.

Repeated irregularities of this kind bear some resemblance to a tremor. Since they occur only when voluntary action is attempted, they have been termed "intention tremor." "Intention tremor" is, however, merely ataxia plus the effort to control it.

The patient, G. C. (National Hospital), at age 22 noticed a weakness in his right instep after walking; two years later the foot dragged. At age 25 he found that he was not able to talk fluently; his words seemed to get tied up with his tongue and he often could not get them out unless he paused; his lips seemed in his way (as if he had "too much lip") and his whole mouth seemed not properly constructed. Somewhat later tremor appeared in his

FIG. 10.—Beginning of "ah" sung by J. H. The record begins with peculiar vibrations. The waves soon become apparently regular in length. Their height increases and diminishes as in a tremolo; this is due to ataxia of breath control.

FIG. 12.—Beginning of "ah" sung by G. C. The peculiar vibrations at the start are very marked.

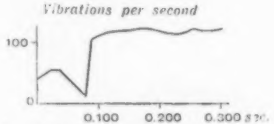


FIG. 13.—Melody plot to fig. 12. After the violent initial jerks the tone becomes quite steady.

FIG. 14.—Portion from the middle of "ah" sung by G. C. The rise and fall of the line indicate variations of the breath (breath ataxia).

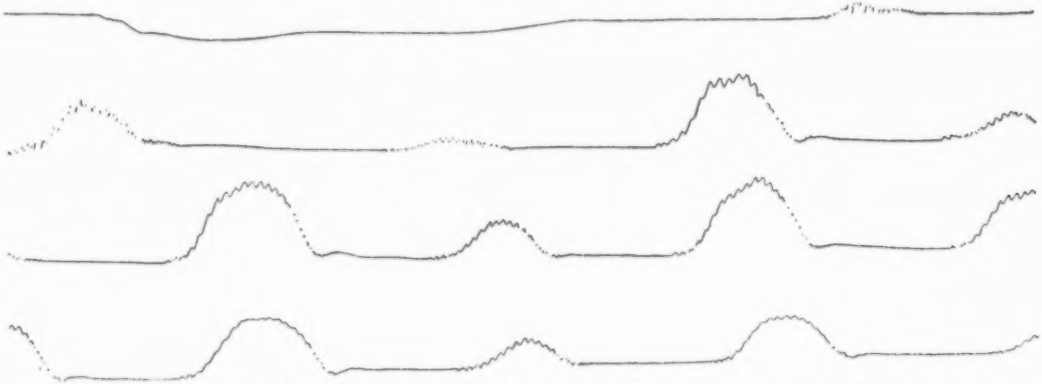


FIG. 15.—Portion of a record of "ah" sung by J. E. Instead of an even series of waves as in fig. 3 the vowel waves appear in pieces that rise and fall quickly like the explosion of "t" in fig. 5. The vowel was in fact composed of a series of pieces shot out separately with silences between them.

hand; his vision was often blurred and double. For the last two years the symptoms have been increasing and extending. Now at age 29 his voice sounds monotonous with laboured enunciation. He often inserts "er" and often lets off some breath at the end of a phrase. On examination his face looked very tired; his walk was irregular; nystagmus was present. The abdominal reflexes were lacking; the knee-jerks were exaggerated and equal; there was no ankle clonus; the plantar reflexes were extensor. In making records he would often halt for a very brief instant before a consonant. Difficult combinations of consonants, like "Peter Piper's peppers," troubled him considerably, but he always practised them, so that unless they were new to him he could speak them fluently.

The peculiar vibrations in G. C.'s records are exemplified by those in fig. 12 for the beginning of "ah" sung. The melody plot is given in fig. 13.

The ataxia in breathing is well shown by the rise and fall of the line in a record of "ah" sung by G. C., reproduced in fig. 14.

At age 14, J. E. (National Hospital), noticed that people sometimes could not understand what he said. Two years later he noticed a difficulty in walking. In the fourth year of his disease he began to shake; in the fifth he was obliged to give up work.

At the age of 24, my observations (by ear) of his speech were summarized as follows: Speed slow, about one-third normal; force increased; vowels normal in length or nearly so; consonants greatly lengthened, three to twelve times; spaces of silence inserted after many consonants and vowels; gasps occasionally inserted; explosions of consonants increased; vowels often brought out as a series of two or more explosions preceded and followed by silence.

In the attempt to sing "ah" the patient emitted a series of explosions with silences between them. A portion of such a record is given in fig. 15. The portions of straight line represent absolute silence and holding of the breath. The sudden and extreme risings in the line register the explosions between silences. The fine waves register the vibrations of the larynx.

Observation of the patient during such an attempt to sing "ah" showed that the muscles of the mouth were held rigid. The abdomen was held in violent contraction without tremor. To resist the resulting pressure either the diaphragm or the glottal lips must have been strongly contracted. As this contraction was relaxed momentarily, the breath burst forth. In addition to all this, the muscles of face, arms and legs were strongly contracted. The violence of the

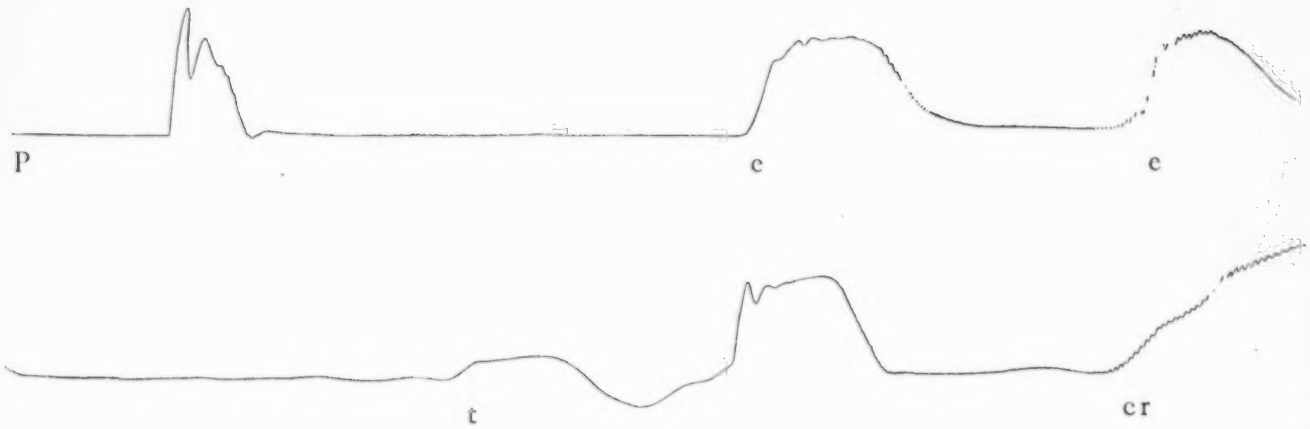


FIG. 16.—Record of "Peter" by J. E. The "P" has a proper explosion (raised line) followed by a few vowel vibrations. It is followed by a long line indicating absolute silence. The vowel "e" is shot out in two pieces. For the "t" there is a wavering outgo and intake of breath followed by an explosion. A silence occurs before the vowel "er" is shot out.

contractions was indicated by the perspiration that was produced and the fatigue of which he complained.

In a word like "Peter" (fig. 16) he closes his lips for the "p" and opens them for its explosion, as shown by the straight line with the rise at the end. The following straight line indicates silence and shows that everything is cramped tight. The vowel "e" is emitted in two explosions followed by a silent cramp. The peculiar rise and fall of the line for "t" shows that instead of the normal silence (occlusion) air was emitted and drawn in; this was due to a coarse jerk of the tongue whereby it could not keep its position firmly across the mouth passage.

Observation of the abdomen during the word "baby" showed that it gave three strong movements. One of these occurred with the explosion of each "b." The third was violent and prolonged after the final vowel. It was evidently produced to cut off the vowel to a normal length; there must have been joint contraction of the diaphragm and abdominal muscles whereby further expulsion of breath was hindered. The patient said he felt he must make this cramp after each word as otherwise he does not feel that the word is finished. He can postpone this final cramp for several seconds, but he does not feel comfortable until he makes it. Instead of ending a word by relaxation as we normally do, he—in order to avoid continued vowel explosions—ends it by a cramp. The action finally becomes a habit.

Silent spaces (cramps) were habitually inserted by him after both vowels and consonants. Including the silent spaces and the explosions, the vowels were much lengthened, the consonants often immensely so. Inspiratory gasps were frequent.

The speech of this patient brings out forcibly two factors that characterize the speech of this disease, namely, ataxia and the effort to control ataxia. For the latter process a special term is needed; "anataxia" conforms to the habits of medical terminology. The speech of this disease, like the stamping gait of tabes, is an expression of ataxia plus anataxia, and not of ataxia alone.

The speech records do not yet afford any direct conclusion whether the ataxia is motor or sensory. The fact that tabetic patients with sensory ataxia complain of loss of sensation as the cause of their irregular movements, while sclerotic patients never do so, seems to justify the general conclusion that the ataxia in disseminated sclerosis is motor.

The speech of this patient also affords a good illustration of the overaction of hypertonic muscles.

The history of P. G. (National Hospital), aged 16, taken in October, 1911, states that about a year previously he had been troubled by mistiness before the eyes, squint and double vision. About eight months later there was unsteadiness of the legs and arms; he walked badly and could not hold a cup. After about eleven months he had been troubled by precipitate micturition. He was described as being very intelligent and having a good memory. All his eye movements were jerky and inco-ordinated; the left external rectus was almost totally lacking in power: there was nystagmus in all directions. His speech was described as slow and stumbling. There was inco-ordination in arms and legs. There was a slight tremor on both sides; this was increased in movement. The gait was unsteady; the Romberg sign was absent. In February, 1914, he had had an "attack" while at work in which he "contorted," lost consciousness and was very sick. At home he had seven or eight fits more, in which he lost consciousness; the convulsions occurred on the right side, involving face, arm and leg. On admission to the National Hospital in March, 1914, he is described as good-natured, stupid, with poor attention and memory. His optic discs were highly coloured; there was a rhythmic moving of the pupils during convergence; there was ample nystagmus of moderate speed. He was affected by double vision. There was weakness in the region of the left facial nerve. His speech was described as dysarthric; the right arm was spastic; both legs were spastic, the right more so. Only feeble movements were possible; there was no incontinence of urine.

Records of P. G.'s speech were made in April, 1914. Upon being told to sing "ah" it was a long time before he started. A breathy, wavering "ah" was produced. With each repetition he made the sound successively shorter, probably from fatigue. Although he was able to make all sounds correctly, he frequently slurred them or left them out in actual speech, thus he said "go' morning" and "hippopot'mus." In repeating the phrase "Peter Piper's peppers" he regularly took a breath before and expelled it after each word, thus:—

↗Peter↘▲ ↗Piper's↘▲ ↗peppers↘▲

An example of elision was seen in—

↗Peter↘▲ ↗Piper↘▲ ↗peppers↘▲

and of attraction in—

↗Peter's↘▲ ↗Piper's↘▲ ↗peppers↘▲

The "s" was often enormously prolonged, as "s-s-s-s-stead, yes-s-s-s, pleas-s-s-s-sure."

The peculiar laryngeal vibrations were constantly present in his records.

These phenomena can be reduced to ataxia and spasticity with the efforts to control them. The initial hesitation is merely to get the muscles under control. The wavering is due to a breath ataxia. The

fatigue is naturally due to the great muscular effort. The slurring and elision result from the excessive efforts at control. The shouting out of a series of words in separate breaths indicates inability to control the breath ataxia for any considerable time. The prolongation of the "s" was presumably due to difficulty in releasing an excessive contraction of the tongue. The peculiar laryngeal vibrations are the direct registration of laryngeal ataxia as explained above.

The shooting out of each word by a separate breath impulse is probably what has given rise to the description "staccato speech." This staccato is evidently the result of an attempt to control the breath ataxia. Although such staccato effects are found in some cases, they are absent in most. Staccato speech indicates disseminated sclerosis—unless further research finds it in other diseases—but it is quite wrong to say that the speech of this disease is characteristically staccato.

One special fact requires explanation, namely, the attraction of "s" in "Peter's Piper's peppers." In general paralysis the cases of attraction, elision, transposition, &c., are attributed to a cortical defect that produces a symptom-group known as "apraxia." I have observed precisely similar cases in epilepsy, paralysis agitans and mental backwardness, where the cause was certainly not the cortical lesion that produces apraxia, but was simply mental dulness or sluggishness. It is impossible to decide at present whether these cases in disseminated sclerosis indicate apraxia or mental degeneration.

The patient, E. A. E., was admitted to the London County Council Asylum at Claybury at the age of 19 on a certificate which stated that "she is continually crying and singing: she imagines she is in a caravan and sees imaginary horses in the ward." On admission her sensations were described as normal, as were also the pupillary reflexes. She was not able to make many movements owing to the loss of power and tone of the whole of the muscles of the body. These were somewhat wasted all over, and no particular group of muscles seemed to have been picked out. Her legs were useless. She could hardly raise or move them in bed. There was marked tremor of the right arm and less of the left. The tremor increased on attempting to drink a glass of water. There was a constant coarse tremor of the head. There was no reflex on stimulation of the sole of the foot. There was increase in the reflex jerks of the muscles of arm and hand, on the left perhaps more markedly than on the right. There was nystagmus on looking every way except downward. She said she was unable to swallow as well as she used to. She was unable to get rid of bronchial secretion easily. Her eyesight was failing. Her memory was fair. Her speech was laboured. She was suffering from mental confusion and was anxious, depressed, and emotional. There was some history of her having been in a hospital for a similar difficulty a year or more ago.

She seemed to be an advanced case of this disease, showing the somewhat unusual features of very rapid progress and great mental disturbance. When I attempted to take records the patient simply asked continually, "Have I a father and mother?" With some difficulty she was induced to speak into the apparatus. After a time she became quite at ease.

She died twelve days after admission. At the autopsy, the following observations were made: There was wasting of the leg muscles, especially on the right. There was no thickening or opacity of the cerebral membranes. Extensive sclerotic areas could be seen in various parts at the base of the brain. The lungs showed areas of broncho-pneumonia.

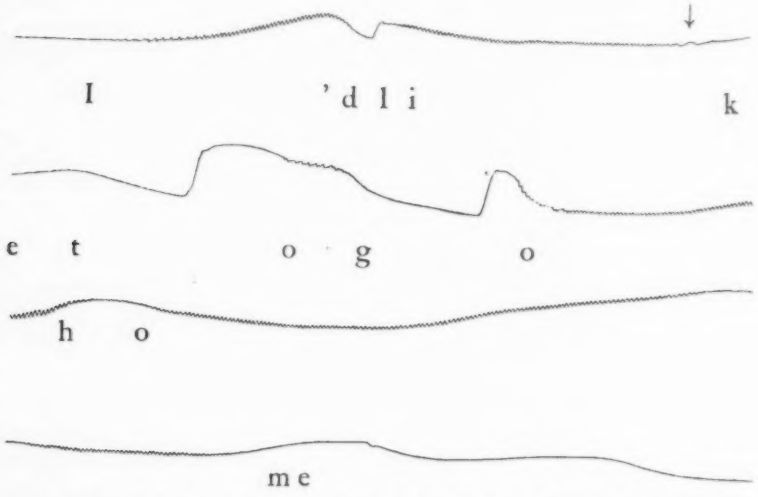


FIG. 17.—Record of "I'd like to go home," by E. A. E. The record is nearly twice as long as a normal one. The "d," "t," and "g," all show their occlusions (descent to the base line) with explosions (sharp rise). The "k" shows a rising line that indicates an incomplete closure of the mouth by the tongue. The vowels are very long. The "m" is lost in a long expulsion of breath.

A record of "I'd like to go home," by E. A. E., is shown in fig. 17. The "I" is breathy (as shown by the rise of the line) and long. The "d" is not a sudden drop in the line as it should be normally, but is a gradual descent; this denotes that the first portion is breathy. The explosion is even more distinct than in a normal record. The "l" is quite distinctly marked. The "i" is long. The slow descent of the line shows that the "k" is fricative instead of occlusive, that is, that the tongue does not completely close the mouth passage. The "k" is quite

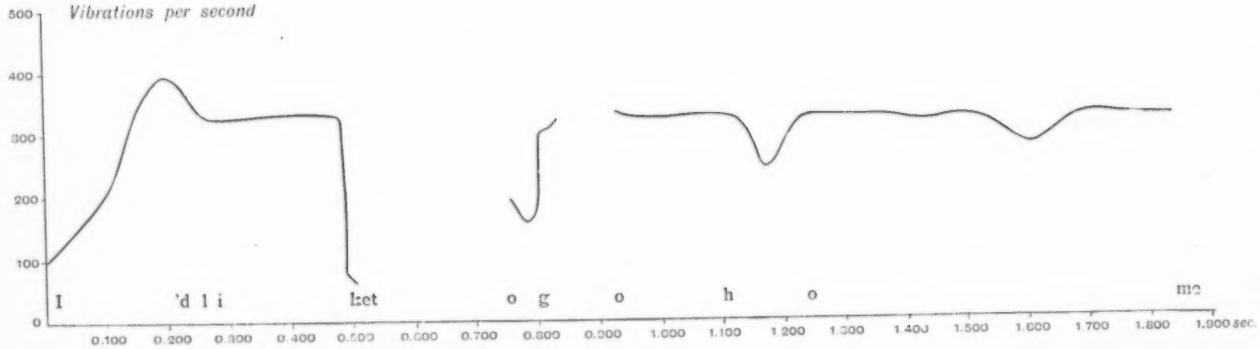


FIG. 18.—Melody plot to fig. 17. The tone rises rapidly from a low pitch of 100 to nearly 400. It finally settles on a pitch of about 325 which is the general pitch of the sentence. At the end of "i" it suddenly drops to a very deep pitch for two vibrations. When it starts again in "o" of "to" it again begins low and rises abruptly. There is some fluctuation in the remainder of the word, but in general the tone is quite monotonous, not even falling at the end.

long; there are peculiar laryngeal vibrations at the beginning, whereas normally there should be none whatever. The "t" shows a fairly good occlusion; it has a strong breathy explosion. The "o" is longer than in the normal record. The "g" has a strong explosion, whereas in this sentence spoken normally it usually has a very faint one or none at all. The "o" thereafter is long. "The "h" is sonant (as indicated by the small vibrations), as it often is normally between two vowels (see fig. 5). The second "o" is enormously lengthened; the rise and fall of the curve show slow variations of intensity. No vibrations could be distinguished in the "m"; it was either surd (i.e., with no laryngeal vibrations) or only faintly sonant.

FIG. 19.—Enlarged waves from fig. 17. Two long peculiar vibrations at the end of "i" in fig. 17 are shown greatly enlarged.

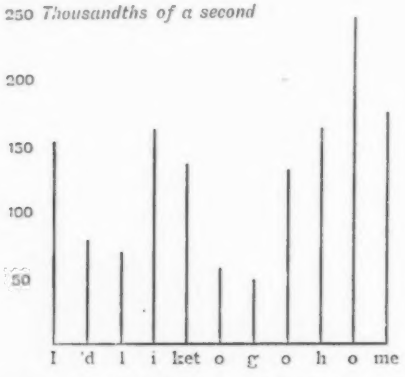


FIG. 20.—Duration Chart to fig. 5. The sounds "I," "i," "o," "h," "o," and "me" are longest; the others are short. The sounds of "k" and "t" cannot be separated.

The melody plot for fig. 17 is given in fig. 18. It shows that the voice starts very low at the beginning and rises rapidly by nearly two octaves. The general course of the melody is monotonous. The small fluctuations of normal melody are absent and the voice does not fall at the end.

The general monotony is presumably the result of spasticity of the laryngeal muscles. The low start and the rise over about two octaves in the initial vowel may be interpreted as due to the difficulty in getting the laryngeal muscles under proper control (ataxia plus anataxia).

At three places the melody plot shows marked interruptions by a fall of sometimes several octaves. Inspection of the original record at these points shows that the series of small regular vibrations is

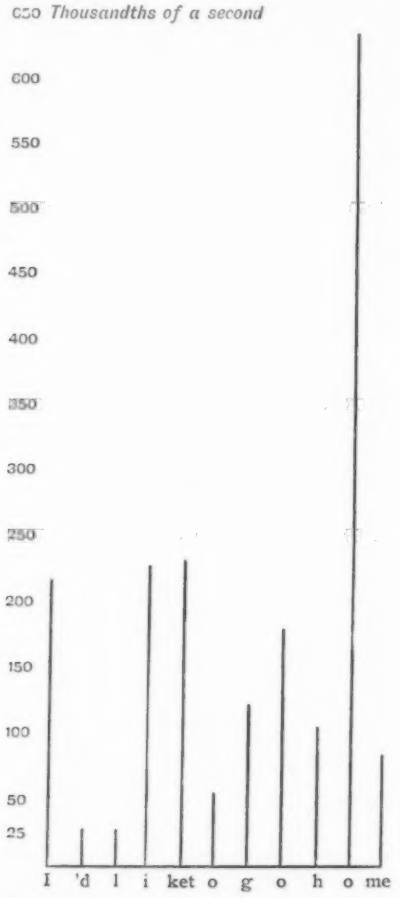


FIG. 21.—Duration Chart to fig. 17. Compared with fig. 20 the sounds of "I," "i," "kt," and the second "o" are slightly lengthened. The third "o" is enormously lengthened. Several of the sounds are shortened. The alteration of duration is quite irregular. It does not proceed on a system of proportionate lengthening as in ordinary slow speech. It does not exaggerate the differences between long and short sounds as in scanning. It does not bring each syllable out in equal times as in staccato speech. It does not separate the syllables or sounds by pauses.

suddenly interrupted by a few large irregular ones. One of these places is shown on an enlarged scale in fig. 19. These are the peculiar irregular vibrations discussed above.

The durations of the separate sounds are obtained by measuring their lengths in the records and turning the results into time by the time equation. The durations for the records in figs. 5 and 17 are as follows :—

	I	'd	l	i	ket	o	g	o	h	o	me.
M. W. F. (normal)	.. 154	.. 77	.. 70	.. 161	.. 137	.. 59	.. 49	.. 132	.. 165	.. 247	.. 176
E. A. E. (dis. scl.)	.. 217	.. 27	.. 26	.. 227	.. 233	.. 51	.. 124	.. 180	.. 106	.. 633	.. 87

As explained above, the " e " of " like " and " home " represents no sound ; the sounds of " k " and " t " are run together.

The results are expressed in the " duration charts " shown in figs. 20 and 21. Comparison of the charts shows : (1) that the sounds in the speech of E. A. E. are not all lengthened proportionately in the normal relations ; (2) that they are not all of one length ; (3) that the lengthening may affect vowels or consonants.

Separated into syllables in the usual way the durations would be as follows :—

	I'd	like	to	go	home.
M. W. F. (normal)	231	.. 300	.. 127	.. 181	.. 588
E. A. E. (dis. scl.)	244	.. 380	.. 177	.. 304	.. 739
Excess of E. A. E. over M. W. F. ..	5 %	.. 27 %	.. 39 %	.. 70 %	.. 26 %

Patients with disseminated sclerosis frequently bring their syllables out in a laboured way that has been called " scanning." Exactly similar speech is often found in hemiplegia, cerebral diplegia, &c.

The term " scanning " is applied in prosody to marking off the long and short or the loud and weak syllables, that is, to indicate the maxima and minima. In scanning a line of verse with the voice, the speaker exaggerates the differences between the two kinds of syllables, making the emphatic syllables more emphatic (longer or louder) and the unemphatic ones less marked (shorter or weaker). This is exactly what the patient with disseminated sclerosis does not do. She does not lengthen the long syllables and shorten the short ones ; she does not give more intensity to the strong ones and less to the weak ones. Moreover she does not do exactly the opposite by making the unemphatic syllables more nearly equal to the emphatic ones, as has often been asserted. Finally, she does not bring each syllable out as a separate unit, as has been asserted by those who claim that the speech is " staccato." She does not even produce the successive efforts at regular intervals. The speech is thus neither scanning nor anti-scanning, nor staccato nor rhythmic.

The records prove clearly that the successive efforts of emphasis show irregular variations in time that indicate a time-ataxia ; there are

also greatly prolonged efforts that are exactly like those of spastic speech. We are justified in concluding that the speech, as far as the durations are concerned, simply shares in the general ataxia and spasticity and that its peculiarities are the results of ataxia and spasticity and the efforts to control them. The term "scanning speech" cannot be used.

§ 4.—Nature of Sclerotic Speech: Diagnostic Table; Early Diagnosis.

From the mass of accumulated records and measurements only typical examples have been reproduced. The conclusions drawn from them were verified for the others. The peculiar vibrations, the monotony, the defective rhythm, &c., were always present.

All these speech signs can be deduced from simple principles. The peculiar vibrations, the irregularities of breathing and the inaccuracy of enunciation are attributable to ataxia. The thickness of speech can be attributed to spasticity, or hypertonia, of the muscles of enunciation. Records and observations of spastic speech (infantile cerebral diplegia, hemiplegia, &c.) show that the condition of hypertonia and the effort to overcome it (anatonia) cause loss of ease of movement, monotony, slowness and alteration of enunciation. All the signs of sclerotic speech recorded up to the present can be explained as the results of ataxia, anataxia, hypertonia and anatonia.

Staccato speech has been found; it is shown to consist of ataxia plus anataxia of breath control. Scanning speech never occurs; when the rhythm of speech is disturbed, the alteration is in the direction of irregularity of rhythm. Pauses occur as the result of breath anataxia; the statement that the syllables are separated by pauses is simply not true. The statement of one authority that there is no characteristic defect for speech in this disease is quite excusable on account of the apparently disconnected and erratic forms in different patients; the records show, however, that they can all be reduced to the fundamental formula.

Owing to the great variations in the symptoms of disseminated sclerosis, it is frequently confused, particularly in its early stages, with other diseases.

The most common confusion is that with hysteria. Although the one consists of organic lesions of the central nervous system and the other is a purely psychical condition, and although the one usually runs an inevitable course where treatment is only palliative, and the

other can be modified or cured by proper mental or hygienic principles, yet at the present day there is frequently no possibility of making a decision as to which disease is present.

Elements	Disseminated sclerosis	Fatigue neurasthenia	General paralysis
I. Speed	Slow	Normal	Normal.
II. Loudness	Normal	Lessened	,,
III. Fatigue	0 or +	+	0
IV. Rhythm :			
(*a*) Duration ..	Lessened	Normal	Normal.
(*b*) Stress	,,	,,	,,
(*c*) Pitch	,,	,,	,,
V. Breath	Irregular	,,	Normal or finely irregular.
VI. Laryngeal tone :			
(*a*) Acoustic character	Clear	Normal or breathy, or tremulous	Normal.
(*b*) Pitch control ..	Irregular	Normal or tremulous	Normal or tremulous.
(*c*) Sentence melody	Monotonous	Normal or melancholy	Normal.
(*d*) Vowel melody ..	Lost in sentence monotony	Normal	,,
VII. Enunciation :			
(*a*) Formation of sounds	Coarse irregularity	Normal or weak	Finely irregular.
(*b*) Co-ordination in duration	,,	Normal	,,
(*c*) Co-ordination in force	,,	,,	,,
VIII. Combination of sounds:			
(*a*) Repetition ..	0	0	+
(*b*) Transposition ..	0	0	+
(*c*) Elision	0	0	+
(*d*) Insertion ..	0	0	+
IX. Ability to change :			
(*a*) Temporarily ..	0	+	0
(*b*) Permanently ..	0	+	0

(I) Speed is given by the length of a word or phrase as compared with the normal length.

(II) Loudness shows itself in larger curves in the records.

(III) Fatigue may show itself in a single word by decrease in loudness, or in a succession of records by cessation of speech, by tremulousness, &c.

(IV) Rhythm depends on relative duration of the syllables (long and short), relative stress (loud and weak) and relative pitch (high and low).

(V) Breathing affects the intensity and regularity of the sounds.

(VI) The laryngeal tone, or tone of the voice, has an acoustic character that is usually clear. Pitch control is shown by the ability to sing tones of different pitch. The sentence melody is the rise and fall of the voice in speaking a phrase or a sentence. The vowel melody is the rise or fall of tone within the individual vowels.

(VII) Enunciation: the formation of sounds requires certain very definite movements of lips, tongue, velum, &c., for each one. For a normal sound the muscular movements must have proper coordination in duration and proper coordination in force.

(VIII) Combination of sounds into words and phrases may be disturbed in the four ways indicated.

(IX) Ability to change. An abnormal mode of speech may be altered temporarily or permanently.

Disseminated sclerosis is also sometimes confused with encephalitis, cerebral-spinal lues, paralysis agitans, general paralysis, pseudo-sclerosis, &c. It is evident that a method would be of value which would definitely decide at the earliest moment whether the trouble is disseminated sclerosis or not. The presence of the peculiar vibrations in a speech record proves the existence of laryngeal ataxia and definitely excludes any of the diseases just mentioned. Laryngeal ataxia occurs in Friedreich's disease and possibly in others yet to be studied. The description of the speech in lenticular degeneration does not seem to indicate anything like that in disseminated sclerosis. I have seen one case that had been diagnosed as lenticular disease; the speech was plainly sclerotic and the diagnosis was a mistake.

The annexed table gives the speech characteristics that are present in disseminated sclerosis when the speech is affected to a noticeable degree. The analyses of two other diseases are added for comparison.

In conclusion it is my most agreeable duty to acknowledge obligations to Dr. F. E. Batten for the use of a consulting room at the National Hospital, for access to many cases with speech defects and for helpful suggestions; to Dr. F. W. Mott and Dr. R. Armstrong-Jones for the Claybury case; and to the National Medical Research Committee for some aid in defraying expenses.

REFERENCES.

For details of the graphic method of recording speech :—

ROUSSELOT. "Principes de la phonétique expérimentale," Paris, 1897.

SCRIPTURE. "Elements of Experimental Phonetics," Yale University Press, 1902, p. 385.

THE FACTORS WHICH GOVERN THE PENETRATION OF ARSENIC (SALVARSAN) AND ANILINE DYES INTO THE BRAIN AND THEIR BEARING UPON THE TREATMENT OF CEREBRAL SYPHILIS.[1]

By JAMES McINTOSH, M.D.,
Late Research Scholar of the Grocers' Company, &c.,

AND

PAUL FILDES, M.B.,
Assistant Bacteriologist to the London Hospital.

(*From the Bacteriological Laboratory of the London Hospital, Professor William Bulloch, F.R.S.*)

In a previous paper [2] we have shown that after intravenous injections of salvarsan and neosalvarsan no arsenic can be found in the brain substance, this result being due not to any lack of affinity between the brain-cells and the drug, but to an obstruction to the passage of the drug from the blood-stream into the brain substance.

In the present paper we detail experiments which we have devised to throw light upon the factors involved in penetration or lack of penetration into the brain. We required for this investigation a group of substances which were relatively non-toxic when injected intravenously and readily recognizable in the tissues after the death of the animal. Various dyes, some of which had already been investigated by Ehrlich, were selected for our purpose.

Our mode of procedure was as follows :—

A rabbit was injected intravenously with a large dose of an aniline dye and killed about five minutes later by air embolism—10 c.c. of air being injected into the ear vein. The animal was then dissected and the distribution of the stain noted in the tissues generally. The brain and cord were next exposed and examined. The cerebrospinal fluid was collected in a capillary pipette by puncture of the membranes after exposure of the cord; by this method the fluid may be obtained

[1] Work carried out under grants from the Medical Research Committee.

macroscopically free from blood. The pipettes were viewed along their long axis in order that small traces of stains might be detected.

By the method of intravenous injection stains may be divided into two groups, namely, those which stain the central nervous system and those which do not.

Group I.—STAINS WHICH COLOUR THE TISSUES GENERALLY AND ALSO THE CENTRAL NERVOUS SYSTEM.

(1) *Methylene Blue.*

This stain was used as an intravital stain by Ehrlich. It is very rapidly reduced to a colourless compound by the tissues, and this fact must be borne in mind when investigating its distribution within the body.

The effect of methylene blue is as follows :—

Experiment 1.—A rabbit (1,300 grm.) was injected intravenously with 1·5 c.c. of a saturated solution of methylene blue ("medicinal pure" (Grübler). The animal immediately became restless and tinged with blue. This coloration rapidly passed off and the symptoms were disappearing when the rabbit was killed by air embolism five minutes after the injection. The internal organs were examined immediately and found to be uncoloured, but the muscles were slightly blue and this became intensified on exposure to air.

The membranes of the brain and cord were uncoloured, the cerebro-spinal fluid was also uncoloured and remained so even after the addition of hydrogen peroxide. The brain was at first colourless, but on exposure to air became distinctly blue ; the cord on the other hand was uncoloured and remained so.

The brain and cord were placed in Bethe's solution [2] for the fixation of methylene blue. The blue colour then became more intense and was seen to be confined to the grey matter of both structures. After fixation and embedding in paraffin sections were cut and the blue staining was found to be diffuse. No intracellular granules were observed and no differential staining of the tissues.

Experiment 2.—In a second experiment a rabbit (2,000 grm.) was injected with a larger dose, 10 c.c., of a saturated solution. Toxic symptoms were more profound, but were passing off when the animal was killed five minutes later.

[2] Dissolve ammonium molybdate (1 grm.) in 10 c.c. of distilled water and add 1 c.c. of hydrogen peroxide—the solution will become yellow—then add one drop of hydrochloric acid.

The appearance of the tissues was similar, although in this case the lungs were distinctly stained.

Thus, after the injection of methylene blue the distribution of the stain in the central nervous system is as follows: Meninges, unstained; cerebrospinal fluid, unstained; grey matter, stained diffusely, no granules; white matter, unstained.

(2) *Neutral Red.*

This stain was also used by Ehrlich for intravital purposes. It has very little toxicity, and is rapidly excreted by the kidneys.

Experiment 3.—A rabbit (1,500 grm.) was injected intravenously with 15 c.c. of a saturated solution of neutral red (Grübler, "for intravital injection"); tetanic spasms came on before the injection was finished and rapidly increased until complete rigidity was present. These symptoms, however, soon began to pass off and the animal was killed ten minutes later. General staining was present externally.

On dissection the organs were found to be stained, the liver and kidneys being dark purple and the muscles bright red.

The meninges of the central nervous system were slightly pink, but the cerebrospinal fluid was entirely colourless. The brain, on the other hand, was intensely stained a dark red, while the cord was pink. Incisions into the brain and cord showed that the colour was confined to the grey matter. Portions were fixed in formalin and embedded in paraffin. In sections the coloration of the grey matter was found to be diffuse, although there appeared to be a slight differential staining of the cortical layer of the ganglion cells. No granules, however, were observed in these cells.

Experiment 4.—In a further experiment a rabbit (1,600 grm.) was injected with 7 c.c. of the saturated solution and an exactly similar distribution of the stain was found. In this case, however, a slight coloration was present in the cerebrospinal fluid due to a small admixture of blood.

Amongst the other stains which affected the grey matter of the central nervous system were alizarin blue and malachite green; while indophenol and aurantia did so only slightly. In no case, however, was the effect quite so marked as with neutral red and methylene blue, perhaps because the less solubility and greater toxicity did not allow of their use in such large doses.

Group II.—STAINS WHICH COLOUR THE TISSUES GENERALLY, BUT NOT THE CENTRAL NERVOUS SYSTEM.

(1) *Fluorescine.*

Experiment 5.—A rabbit (1,500 grm.) was injected intravenously with 5 c.c. of an alkaline watery solution of fluorescine (1 per cent.). No toxic symptoms followed and the animal, distinctly yellow, was killed after five minutes by air embolism.

On examination the internal organs were slightly yellow, while the kidneys were intensely stained. The muscles were golden yellow.

On exposing the central nervous system, the meninges were found to be stained but the cerebrospinal fluid was quite colourless. Both the brain and the spinal cord were entirely free from colour.

Experiment 6.—Another rabbit (1,500 grm.) was injected with 10 c.c. of the same solution of fluorescine. Exactly similar results were obtained as in the last experiment, that is, the brain and cord were unstained while the meninges were stained. In the cerebrospinal fluid a trace of fluorescine was detected, due, no doubt, to the presence of a slight quantity of blood which could be detected by the microscope.

(2) *Indigo-carmine.*

Experiment 7.—A rabbit (1,800 grm.) was injected with 20 c.c. of a saturated solution of indigo-carmine in saline solution. No toxic symptoms appeared and the animal became tinged green. In five minutes it was killed.

The internal organs and muscles were stained green. The meninges of the brain and cord were distinctly coloured, but the cerebrospinal fluid and the nerve tissue were entirely unstained.

Other stains which did not colour the central nervous system were found to be acid fuchsin, light green, trypan red, trypan blue, and pyrrhol blue.

From our experiments, a number of which have been given above, it is clear that the aniline dyes tested may be classified according as to whether, on intravenous injection, they stain the nervous tissues or not, and an examination of the physical properties of these substances might reveal the factors by virtue of which one group stains while the other does not.

From analogy with our experiments with salvarsan we argued that one stain did not colour the brain, while another did, because the former

did not penetrate. The blood capillaries of the brain are peculiar when compared with those of other parts of the body, in that they are surrounded by an extra adventitial sheath, and we supposed that this extra sheath might act as a barrier to certain drugs while permitting the passage of others. We therefore investigated the chemical formulæ of these stains, but were unable to find any atomic arrangement which was characteristic of the two groups.

Similarly the diffusibility of the stains did not correspond with their staining reactions.

The question of solubility appeared to be of more importance. Ehrlich [1] stated that neurotropic substances are lipotropic, and Overton [3] that before a substance could penetrate into a cell it must be soluble in the cell membrane, and these membranes being lipoids it follows that such a substance must be soluble in lipoid solvents such as alcohol and ether. The solubility of the dyes which we used in alcohol and ether did not correspond with their staining reactions, but when we used chloroform as a solvent we found that those stains which coloured the brain were soluble in chloroform, while those which did not were insoluble. It would appear, therefore, that the solubility in chloroform or related substances is an important factor in determining a passage from the blood into the nervous tissues or not.

On applying this test to salvarsan we found that neither salvarsan nor neosalvarsan were soluble in chloroform; we are, therefore, inclined to believe that this fact can account for their absence from the brain.

In order to test this suggestion we have examined a large number of organic and inorganic arsenical compounds, for the majority of which we have to thank Miss F. M. G. Micklethwait, Imperial College of Science. No very striking result was obtained, due no doubt to the fact that hardly any of these compounds were soluble in chloroform, and the few which were, were either completely insoluble in water or immediately hydrolysed by it. Certain combinations, however, of arsenic and aniline colour bases gave encouraging results, and after their administration arsenic was demonstrable in the brain, but owing to the War the work had necessarily to be suspended in this interesting stage.

Conclusions.

(1) Certain dye substances can pass directly from the blood to the brain substance proper without being found in the cerebrospinal fluid, while others fail to penetrate into the brain.

(2) The chief factor which governs the passage of the dyes is their solubility reactions.

(3) This is a peculiar solubility and not a general lipoid solubility.

(4) It corresponds to a solubility in chloroform and in water or perhaps to their partition coefficient in these liquids.

(5) The present-day arsenical remedies are, to some extent, inefficient in the treatment of syphilis of the central nervous system because they do not possess the necessary solubility to allow them to pass from the blood-vessels into the brain substance. Their relative inefficiency has nothing to do with their absence from the cerebrospinal fluid.

LITERATURE.

[1] Ehrlich, P. *Therap. Monatshefte*, 1887, Bd. i, S. 90.
[2] McIntosh, J., and Fildes, P. *Proc. Roy. Soc.*, B., 1914, vol. lxxxviii, p. 320.
[3] Overton, E. *Jahrb. wiss. Bot.*, 1899, Bd. xxxiv, S. 669.

INDEX

TO

BRAIN:

A JOURNAL OF NEUROLOGY,

FOR

VOL. XXXIX, 1916.

INDEX OF AUTHORS.

Adrian, E. D. PAGE
The electrical reactions of muscles before and after nerve injury 1

Batten, F. E.
Acute poliomyelitis 115

Brown, T. Graham, and Stewart, R. M.
On disturbances of the localization and discrimination of sensations in cases of cerebral lesions, and on the possibility of recovery after a process of training .. 348

Fearnsides, E. G.
Intracranial aneurysms 224

Fildes, Paul, and McIntosh, J.
The factors which govern the penetration of arsenic (salvarsan) and aniline dyes into the brain, and their bearing upon the treatment of cerebral syphilis .. 478

Halliburton, W. D.
The possible functions of the cerebrospinal fluid 213

Holmes, Gordon, and Lister, W. T.
Disturbances of vision from cerebral lesions, with special reference to the cortical representation of the macula 34

Krabbe, K.
A new familial infantile form of diffuse brain-sclerosis 74

Lister, W. T., and Holmes, Gordon.
Disturbances of vision from cerebral lesions, with special reference to the cortica representation of the macula 34

McIntosh, J., and Fildes, Paul.
The factors which govern the penetration of arsenic (salvarsan) and aniline dyes into the brain, and their bearing upon the treatment of cerebral syphilis .. 478

Roberts, F.
Degeneration of muscle following nerve injury 297

Scripture, E. W.
Records of speech in disseminated sclerosis 455

Stewart, R. M., and Brown, T. Graham.
On disturbances of the localization and discrimination of sensations in cases of cerebral lesions, and on the possibility of recovery after a process of training .. 348

INDEX OF SUBJECTS.

PAGE

Acute poliomyelitis. F. E. Batten 115

Aneurysms, intracranial. E. G. Fearnsides 224

Aniline Dyes, the factors which govern the penetration of arsenic (salvarsan) and, into the brain, and their bearing upon the treatment of cerebral syphilis. J. McIntosh and Paul Fildes 478

Arsenic, the factors which govern the penetration of, and aniline dyes into the brain, and their bearing upon the treatment of cerebral syphilis. J. McIntosh and Paul Fildes 478

Brain-sclerosis, a new familial infantile form of diffuse. K. Krabbe 74

Cerebral lesions, disturbances of vision from, with special reference to the cortical representation of the macula. Gordon Holmes and W. T. Lister 34

disturbances of localization and discrimination in cases of, and on the possibility of recovery of these functions after a process of training. T. Graham Brown and R. M. Stewart 348

Cerebrospinal fluid, the possible functions of the. W. D. Halliburton 213

Disseminated sclerosis, records of speech in. E. W. Scripture 455

Disturbances of vision from cerebral lesions with special reference to the cortical representation of the macula. Gordon Holmes and W. T. Lister 34

Electrical reactions, degeneration of muscle following nerve injury. F. Roberts .. 297

the, before and after nerve injury. E. D. Adrian 1

Familial: a new familial infantile form of diffuse brain-sclerosis. K. Krabbe .. 74

Intracranial aneurysms. E. G. Fearnsides 224

Macula, disturbances of vision from cerebral lesions, with special reference to the cortical representation of the. Gordon Holmes and W. T. Lister 34

Muscle, degeneration of, following nerve injury. F. Roberts 297

Muscles, the electrical reactions of, before and after nerve injury. E. D. Adrian .. 1

Nerve injury, degeneration of muscle following. F. Roberts 297

the electrical reactions of muscles before and after. E. D. Adrian .. 1

Poliomyelitis, acute. F. E. Batten 115

Records of speech in disseminated sclerosis. E. W. Scripture 455

Recovery of sensation, disturbances of localization and discrimination in cases of cerebral lesions and on the possibility of recovery of these functions after a process of training. T. Graham Brown and R. M. Stewart 348

PAGE

Salvarsan, the factors which govern the penetration of arsenic (salvarsan) and aniline dyes into the brain, and their bearing upon the treatment of cerebral syphilis. J. McIntosh and Paul Fildes 478

Sclerosis, brain, a new familial infantile form of diffuse brain-sclerosis. K. Krabbe .. 74

Sensation, disturbances of the localization and discrimination of sensations in cases of cerebral lesions, and on the possibility of recovery of these functions after a process of training. T. Graham Brown and R. M. Stewart 348

Speech, records of, in disseminated sclerosis. E. W. Scripture 455

Syphilis, cerebral, the factors which govern the penetration of arsenic (salvarsan) and aniline dyes into the brain, and their bearing upon the treatment of. J. McIntosh and Paul Fildes. 478

Vision, disturbances of, from cerebral lesions, with special reference to the cortical representation of the macula. Gordon Holmes and W. T. Lister 34